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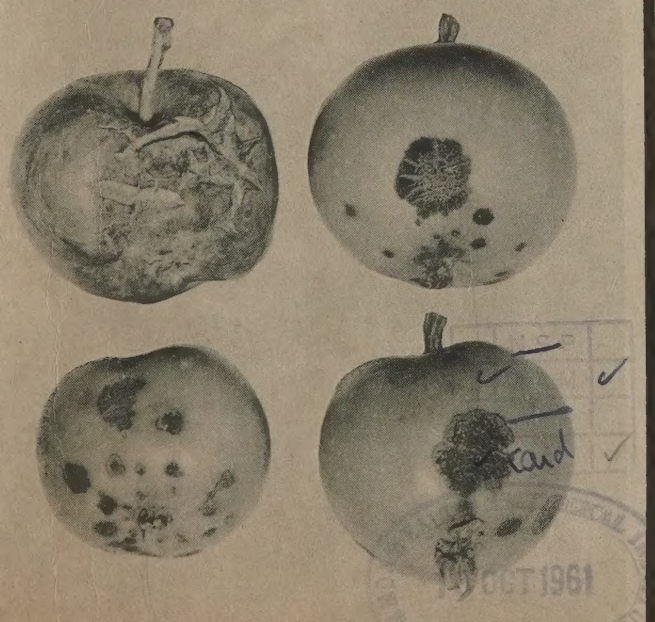

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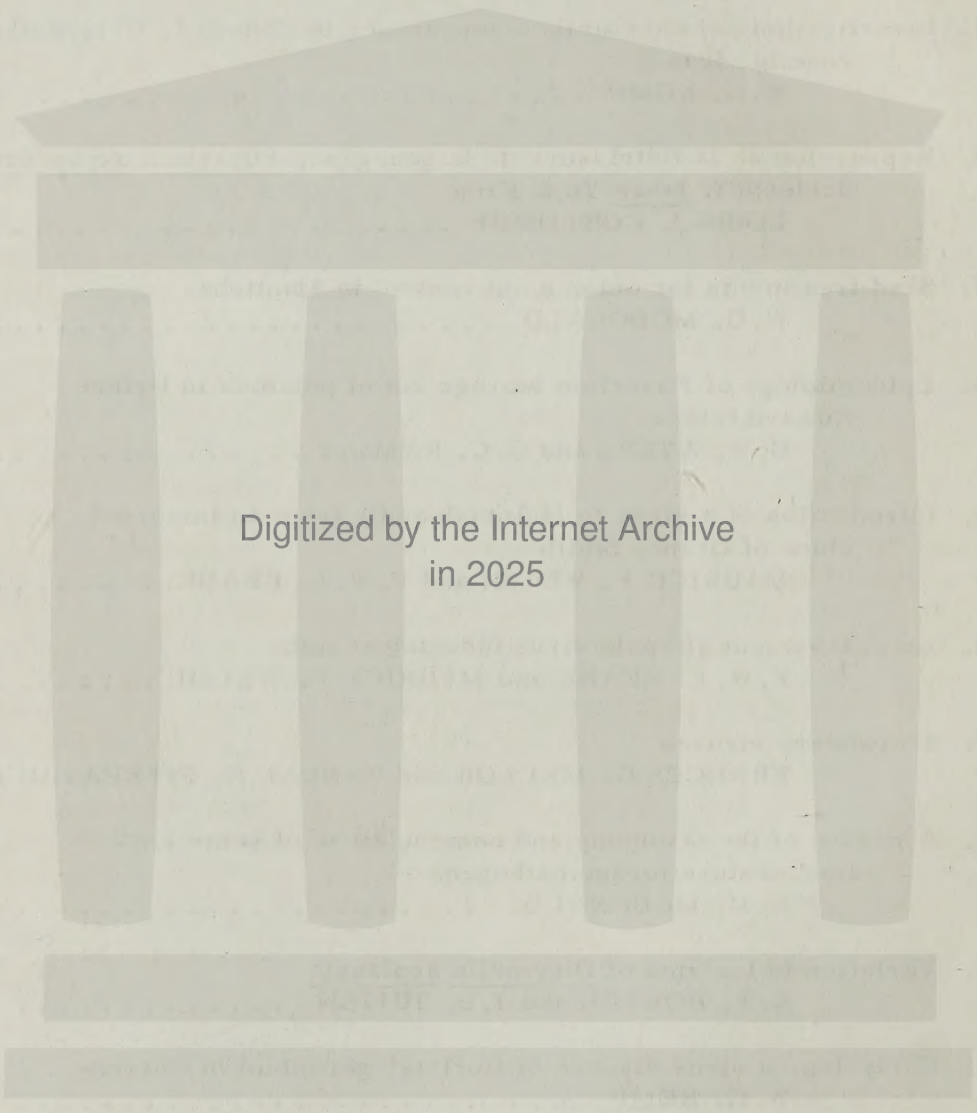

INVESTIGATIONS OF CHRYSANTHEMUM VIRUSES IN CANADA

I. Chrysanthemum Rosette Virus[1]

W. G. Kemp[2]

Abstract

A virus disease of chrysanthemum, hitherto unreported in Canada, is described. The causal agent is considered to be identical with, or closely related to the Ivory Seagull rosette virus. It has been transmitted from symptomless plants of the variety Wilson's White to Blazing Gold by grafting and mechanical inoculation where it causes veinbanding, leaf distortion and dwarfing, and later, rosetting of the new growth. This virus has been detected in two additional florists' chrysanthemum varieties and in nine garden varieties.

Introduction

In 1959, during the routine graft-indexing of florists' chrysanthemum varieties to select plants free from stunt virus for further development and production, a commercial propagator in Southern Ontario observed unfamiliar symptoms in Blazing Gold which had been grafted to symptomless plants of the variety Wilson's White. The grafted plants that showed unusual symptoms suggestive of a virus disease and rooted cuttings from affected stock of this variety were submitted to the Research Laboratory, Vineland Station, Ontario for a diagnosis of the condition.

Only four virus diseases of Chrysanthemum morifolium Ramat. have been reported in Canada: aster yellows (6); spotted wilt (5); stunt (1); and stunt-mottle (7). None of the viruses that cause these diseases produce symptoms in the indicator variety Blazing Gold that are identical with those induced by the virus detected in plants of Wilson's White. Consequently, further investigation of this graft-transmissible disorder was conducted. This paper records the identity of the causative agent and presents additional data relative to its transmission and its occurrence.

Materials and Methods

Plants of the variety Wilson's White carrying the unknown virus (WWV), without symptoms, have been used as the source of the virus throughout this investigation. These stock plants were further assayed for the presence of other chrysanthemum viruses by a graft-inoculation method commonly used by specialist propagators to select virus-free material. Healthy scions of test

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varieties approximately four inches long were cleft-grafted to succulent, two- to three-inch stem sections of the plants to be indexed. The grafts were then bound tightly with surgical latex strips, and immediately stuck in two-and-a-half-inch clay pots filled with a peat-perlite mixture to root. When rooted, these grafts were repotted in four-inch pots, staked, and grown to flowering. Either Bronze or Pink Mistletoe chrysanthemums were used to detect the stunt, aster yellows and mosaic viruses. The variety Blazing Gold was used to indicate the presence of both the flower distortion and the rosette viruses.

Sap inoculations to Nicotiana tabacum L. var. Harrow Velvet and to Petunia hybrida Vilm. were used to detect aspermy virus and to further help in the identification of the so-called mosaic viruses. Mechanical inoculations were made by macerating infected chrysanthemum leaf tissue in its own weight of distilled water or 0.1 M phosphate buffer at pH 7.0. Carborundum was dusted lightly over the foliage of the indicator plants prior to rubbing them gently with the forefinger moistened with inoculum.

Symptoms

WWV-infected Wilson's White rarely shows detectable disease symptoms which, even when present, are of uncertain diagnostic value because infected plants may be carrying other viruses as well. For the most part, this variety has remained symptomless or shown mild, transitory vein clearing during the 18-month period that the originally infected stock plants have been under surveillance. No abnormal flowers were observed nor were they reported by the propagator from which the material came.

In graft-inoculated Blazing Gold, WWV initially produces pronounced veinbanding, leaf distortion and puckering in young plants. Later, growth is checked and subsequent development is rosetted. Stems of stunted plants show extremely short internodes and the rosetted foliage is noticeably dwarfed (Fig. 1 A-B). Symptomless regrowth of severely rosetted Blazing Gold sometimes occurs, particularly in the latter part of the summer. Flowers on the infected indicator variety are slightly misshapen. No distinctive symptoms occurred in three to five months on the graft-inoculated Bronze and Pink Mistletoe varieties.

Transmission

By grafting: The first successful transmission of WWV was observed in June, 1959, on four Blazing Gold scions that were top-grafted to four symptomless but different Wilson's White sources during the previous March.

Subsequently, in March, April, June, and August, 1960, a series of top-grafts were made using as stock, sources of the variety Wilson's White known to be infected with WWV and as scion, the varieties Blazing Gold and Pink Mistletoe. Twenty-four of the 30 Blazing Gold - Wilson's White graft combinations that were successfully rooted and grown, showed rosetted growth in two months. None of the scions of the 11 rooted Mistletoe-Wilson's White combinations showed symptoms in three to four months.

Fig. 1. Symptoms of chrysanthemum rosette virus.

A. Blazing Gold showing leaf distortion and rosetting three months after graft-inoculation.

B. Left, Blazing Gold top-grafted to rosette infected Wilson's White.
Right, Blazing Gold top-grafted to rosette-free Wilson's White.

Transmission was detected on some of the Blazing Gold scions as early as five weeks after the March, 1960, grafts were made. Symptoms were apparent four weeks after the April graftings and six weeks after the June and August graft-inoculations. The initial symptoms appeared as a clearing of the larger veins followed by puckering and distortion of the youngest leaves. All of the infected scions later showed stunting, rosetting, and leaf dwarfing.

By sap: Attempts were made to effect transmission of the virus by mechanical inoculations with extracts from infected chrysanthemum plants. Sap inoculations in June, 1959, March, July, and August, 1960, failed to demonstrate virus transmission to petunia or tobacco seedlings from WWV-infected leaves in a six-week period. Leaf extracts from a few of the inoculated tobacco and petunia plants did not induce rosette symtoms in back-tests to Blazing Gold.

In July, August, and October, 1960, sap from infected Blazing Gold leaves was rubbed on the foliage of 10 young four-to six-inch plants of Blazing Gold and Pink Mistletoe. In seven to eight weeks after the July inoculation dates, two of the 10 Blazing Gold became infected. No additional plants of this series showed symptoms after three months. Neither indicator variety showed symptoms after four months following the August inoculations. In the October test, one of the 10 inoculated Blazing Gold plants became infected in five weeks; another showed visible symptoms during the sixth week. At that time, a six-inch terminal portion of the stem of each of the eight plants that were symptomless was removed with a sterile scalpel. Two of these topped plants became infected; the first two weeks later, the other in the third week.

No increase in the number of plants infected was obtained by extracting the sap from infected chrysanthemum leaves with 0.1 M phosphate buffers adjusted to pH 5.0, 6.0, 7.0 and 8.0.

By insects: Apterous Myzus persicae Sulz. (Homoptera:Aphididae) failed to transmit the virus from infected Blazing Gold to healthy Blazing Gold. During the summer of 1960, nonviruliferous aphids were reared on healthy radish seedlings prior to starving them for from four to five hours on moist filter paper in a petri dish. They were then given an acquisition feed lasting from two to five minutes on detached leaves of infected Blazing Gold. Aphids were then transferred to each of 10 young, healthy Blazing Gold indicator plants and these plants were caged immediately. Twenty-four hours later, all of the cages were removed and the plants were sprayed with malathion to destroy the aphids. No transmission occurred in three months in either of the two tests conducted in July and August. Healthy control plants did not become infected following the transfer to them of Myzus persicae previously fed on healthy detached Blazing Gold leaves.

Two additional aphid species, Macrosiphoniella sanborni (Gill.) and Aphis sp., found to breed well on chrysanthemums, were used in other transmission tests. Single WWV-infected Blazing Gold plants, each previously infested with one of the aphid species, were caged with six healthy indicator

plants. The healthy plants were slowly colonized by alatae of each species from the infected plants. After a month, all of the plants were removed from each cage and sprayed with malathion. At the time of their removal from the cages, no symptoms were apparent on the original healthy Blazing Gold nor were they observed on these same plants three months later.

Identity of the Virus

WWV produces certain symptoms in the chrysanthemum indicator variety Blazing Gold in common with the flower distortion virus, the aster yellows virus, and the rosette viruses. Brierley and Smith (3) found that Blazing Gold showed a characteristic rosetted growth when infected with flower distortion virus at all seasons and that this variety responded to inoculation with aster yellows virus in a similar manner in winter but not in summer.

However, distinct differences exist between WWV and either of the other two viruses that exclude the possibility of any relationship. Flower distortion virus is not transferred mechanically whereas WWV is; it also induces symptoms in Blazing Gold a month after WWV can be detected in this variety. Furthermore, the distortion virus is reported to be lethal to Blazing Gold between five and seven months after graft-inoculation (3). No lethal effects have been noted in Blazing Gold-Wilson's White graft combinations after an eight month period. The failure of aster yellows virus to transfer mechanically and the fact that it usually induces green flowers in infected varieties indicates that it is not similar to WWV, which can be transferred by sap inoculations and has not produced green flowers in either Blazing Gold or Wilson's White.

WWV, however, appears to be identical with, or closely related to, Brierley and Smith's (4) Ivory Seagull rosette virus. It is one of two additional viruses known to occur in the United States that induces a rosetting effect in Blazing Gold at all seasons except in summer. Both WWV and Ivory Seagull rosette virus are sap-transmissible with difficulty and produce symptoms in Blazing Gold in a month. They have failed to infect petunia following mechanical inoculation and neither has been transferred by Myzus persicae from infected Wilson's White to Blazing Gold in the non-persistent manner. On the other hand, Ivory Seagull rosette virus causes veinbanding with leaf distortion in Golden Mistletoe in from three to four months and rosetting in seven months. WWV has failed to induce visible symptoms in this variety. This, however, may simply be due to the fact that the grafted and mechanically-inoculated plants were under observation for only three months.

The fact that Yellow Rayonante rosette virus, considered by Brierley and Smith (4) to be distinct from Ivory Seagull rosette virus, produces veinbanding, interveinal mosaic, leaf dwarfing, and distortion in Mistletoe one month after grafting and leaf necrosis in two months precludes the possibility of a relationship between it and WWV.

Because of the low percentage of successful mechanical transfers of WWV to the susceptible variety, Blazing Gold, experiments have not as yet been conducted to determine the physical properties of this virus.

Incidence

The distribution of the rosette virus in the large numbers of florists' chrysanthemum varieties that are available commercially in Ontario and its prevalence in an infected variety have not been investigated. Its identification by symptom expression alone is probably inaccurate. The problem of diagnosis is complicated by the almost total absence of symptoms in the leaves of some varieties and by mixed virus infections. Graft-inoculation to Blazing Gold followed by back-inoculation with sap from rosetted plants to the same variety is the most reliable detection method. By this method, the virus has been found to occur alone in Wilson's White and with other viruses in Mason's Bronze and Garnet King as well as in the garden varieties Champion Cushion, Jess Williams, Joan Helen, Lavender Lady, Masquerade, Orsona, Powder-puff, Remembrance, and Reflection.

Discussion

Rosette has not been previously reported in chrysanthemums in Canada and its importance to the crop is difficult to assess. Brierley and Smith (2, 4), who first described the disease in the variety Ivory Seagull in 1950 in the United States, reported that because no specific test was known for detecting this rosette virus in the presence of more infectious viruses, its distribution and and prevalence remain obscure. From the experimental evidence obtained to date, it is unlikely that this disease will be a serious economic factor in chrysanthemum production because of its mild effect on varieties naturally infected with the virus and because of the difficulty of sap transmission. However, though the virus and certain affected varieties appear compatible and the plants produce vigorous growth and quality flowers, the possibility exists that other valuable varieties will be less tolerant and may react in a manner similar to the indicator variety Blazing Gold, or even more drastically.

Acknowledgments

The author gratefully acknowledges the identification of the aphids used in the transmission studies by Mr. W. R. Richards, Canada Department of Agriculture, Research Branch, Entomology Research Institute, Ottawa, Ontario, and the technical assistance of Mrs. P. Heald and Mr. W. Haigh. Thanks are due to Atkin's Flowers Ltd., Leamington, Ontario for providing certain chrysanthemum varieties.

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REPRESSION DE LA FLÉTRISSURE FUSARIENNE DE LA GOURGANE, FUSARIUM OXYSPORUM SCHLECHT F. FABAE YU & FANG[1]

Louis J. Coulombe[2]

Abstract

Experiments were carried out to evaluate 3 different methods for controlling Fusarium wilt of Broad beans. Seed disinfection with Fermate (ferbam), Semesan, Spergon (chloranil) and Arasan (thiram) proved inefficient. However when onion, carrot, rutabaga, tomato, cabbage, beet and lettuce are grown four consecutive years, on a light soil infected with Fusarium oxysporum f. fabae, the percentage of the disease is reduced to nil. Two fumigants, Mylone and Vapam, have reduced the disease to a very low percentage.

Il y a déjà quelques années, nous avons reçu des spécimens de gourganes atteintes de flétrissement fusarien dû à Fusarium oxysporum Schlecht. f. fabae Yu & Fang (1). Au cours d'enquêtes dans les comtés de Charlevois, Chicoutimi et Lac St-Jean, le flétrissement fusarien de la gourgane a été observé dans tous ces comtés sans toutefois causer de dommages très appréciables. Nous avons entrepris des expériences afin de trouver un mode de répression efficace, facile d'emploi et économique. Yu and Fang (2) croient que le pathogène peut vivre au moins trois ans sur les débris de racines malades enfouis dans le sol. Ils ajoutent également que le champignon peut à l'occasion s'attaquer à d'autres légumineuses comme les pois et les vesses. Selon eux, les spores de l'organisme se rencontrent sur la graine. Ces quelques indications nous incitèrent d'abord à utiliser la rotation des cultures, puis la désinfection de la graine et, plus tard, la fumigation du sol comme moyens de répression de la maladie.

Matériel et méthode

La variété de gourgane Windsor a été utilisée.

Les expériences ont été faites sur un sol sablonneux bien pourvu de matière organique. Au début de chaque expérience, le sol a été inoculé abondamment avec l'organisme pathogène cultivé sur un mélange de blé et d'avoine.

Le terme rotation est utilisé ici dans un sens plutôt large. Nous voulions étudier la persistance du pathogène dans le sol et l'influence sur celle-ci des diverses cultures légumières employées en l'absence de la gourgane.

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[2] Phytopathologiste.

Tableau 1. Effet de la rotation sur la persistance de la flétrissure fusarienne de la gourgane

Légumes	Pourcentage de plantes malades après					
	1ère année	2ième année	3ième année	4ième année	5ième année	6ième année
	1955	1956	1957	1958	1959	1960
Témoin	78	76.0	69	73.0	66.0	68.0
Haricot	69	63	60	26.0	12.0	7.0
Oignon	76	61	30	5.0	0	0
Soja	68	64	50	15.0	7.0	6.0
Carotte	68	57	28	13	0	0
Rutabaga	63	57	27	14	0	0
Pois vert	74	68	66	22	16	8.0
Tomate	76	58	30	2	0	0
Chou	72	60	31	5	0	0
Betterave	70	59	26	4	0	0
Laitue	75	60	29	5	0	0

Tableau 2. Effet de la fumigation du sol sur la flétrissure fusarienne de la gourgane

Traitement	Pourcentage de plantes malades			Moyenne
	1958	1959	1960	
Témoin	72.0	66.0	61.0	66.0
Vapam	3.3	1.3	0.9	1.8
Témoin	69.0	68.0	63.0	66.0
Mylone	3.1	2.0	1.3	2.1

Les légumes semés en rotation furent le haricot, l'oignon, le soja, la carotte, le rutabaga, le pois vert, la tomate, le chou, la betterave et la laitue.

L'expérience sur la désinfection de la graine, comme moyen de réprimer la maladie, a été poursuivie pendant trois ans. Les désinfectants utilisés furent le Fermante, le Semesan, le Spergon et l'Arasan.

Cette expérience n'a donné que des résultats négatifs; elle fut alors remplacée par la fumigation du sol comme moyen de répression du flétrissement fusarien de la gourgane. A cette fin, nous avons employé le Vapam et le Mylone.

Résultats

Toute espèce de rotation des cultures se révèle un moyen efficace de répression du flétrissement fusarien sur les sols légers. Les résultats consignés dans le tableau 1 indiquent cependant que lorsque des légumineuses, comme les haricots, les pois et les sojas, font partie de la rotation, la destruction du champignon dans le sol s'opère plus lentement. Il semblerait que l'organisme, sans s'attaquer directement à ces légumineuses, s'accommoderait des détritus laissés dans le sol par les racines de ces plantes. A toute fin pratique, il faudrait éviter de faire suivre les gourganes, dans la rotation, par l'une ou l'autre de ces légumineuses. Il semble qu'une rotation minimum de quatre ans soit nécessaire pour obtenir des récoltes saines.

Par contre, la désinfection des graines ne s'est pas montrée un moyen efficace de répression du flétrissement fusarien de la gourgane. La désinfection a diminué quelque peu le pourcentage des plantes affectées mais elle a eu surtout pour effet de retarder de quelques jours l'apparition de la maladie dans les parcelles traitées.

Au contraire, la fumigation du sol apparaît comme un moyen efficace de répression. Elle a réduit le nombre des plantes malades à un très faible pourcentage (Cf. tableau 2).

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SEED TREATMENTS FOR ONION SMUT CONTROL IN MANITOBA[1]

W. C. McDonald[2]

Abstract

Captan, 4 products containing thiram, and 11 products containing hexachlorobenzene were compared in the field and greenhouse for their ability to control onion smut and for phytotoxicity to onions sown thickly for set production. Anticarie 80, Ortho 80, Captan 50-W, Arasan, Panoram D-31, and Delsan A-D, when pelleted with the seed, were the most effective. Captan and thiram preparations plus 50% heptachlor were safe and effective whereas only one hexachlorobenzene preparation was both effective and safe.

Introduction

Recommendations for the control of onion smut, Urocystis cepulae Frost (U. colchici (Schlecht.) Rabenh.), by seed treatment fungicides differ in various parts of the United States. In New York, Arasan applied to seed previously coated with a methocel sticker gave good control in onions sown at a low rate to produce bulbs (4). Larson and Walker (3) obtained better control with Arasan in Wisconsin, when the seed was sown at a heavy rate to produce sets, by applying the chemical to dry seed rather than to methocel-moistened seed. Arasan and Captan pelleted with the seed increased yields on smut infested land in Minnesota (1). In 1959, Duran and Fischer (2) reported that in eastern Washington Anticarie 80 (80% hexachlorobenzene) pelleted with onion seed was more effective against smut than either Arasan or Captan. Some formulations of 40% hexachlorobenzene were phytotoxic.

Losses from smut occur annually in the onion crops grown in the Winnipeg area. Little effort has been made to control the disease as sufficient smut-free land has been available when fields become badly infested. The acreage sown to onions for set production has increased recently, however, and more interest has been shown in seed-treatment fungicides. The experiments reported here were conducted to determine the effectiveness of Captan and Arasan in controlling smut in southern Manitoba and to compare the phytotoxicity and smut-control properties of products containing hexachlorobenzene and available in Canada.

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[2] Plant Pathologist, Plant Pathology Section.

Materials and Methods

The following seed treatment fungicides were used:

Captan 50-W -- 50% captan. Stauffer Chemical Co. of Canada Ltd. Vancouver, B. C.
Arasan 75 -- 75% thiram. E. I. du Pont de Nemours & Co. Wilmington, Del.
Delsan A-D -- 60% thiram and 15% dieldrin. E. I. du Pont de Nemours & Co. Wilmington, Del.
Panoram D-31 -- 56.2% thiram and 18.8% dieldrin. Panogen Inc. Ringwood, Ill.
Heptachlor-Thiram -- Experimental sample, composition unknown. Green Cross Insecticides. Montreal, Que.
Anticarie 80 -- 80% hexachlorobenzene. H. P. Rossiger Co., Inc. New York, N. Y.
Anticarie 80 -- 80% hexachlorobenzene. French Dyestuffs Ltd. Hamilton, Ont.
Ortho 80 -- 80% hexachlorobenzene. California Spray-Chemical Corp. Maryland Heights, Miss.
HCB 100 -- 100% hexachlorobenzene. French Dyestuffs Ltd. Hamilton, Ont.
Bunt-no-more -- 40% hexachlorobenzene. Green Cross Products. Winnipeg, Man.
No Bunt -- 40% hexachlorobenzene. Chipman Chemicals Ltd. Winnipeg, Man.
Co-op Hexa -- 40% hexachlorobenzene. Interprovincial Co-operatives Ltd. Winnipeg, Man.
Anticarie 40 -- 40% hexachlorobenzene. H. P. Rossiger Co., Inc. New York, N. Y.
Shell Aldrin-Hexachlorobenzene -- Experimental sample, composition unknown. Shell Oil Co. of Canada. Toronto, Ont.
Dual Purpose Bunt-no-more -- 13% hexachlorobenzene and 40% heptachlor. Green Cross Products. Winnipeg, Man.
Sanocide -- 40% hexachlorobenzene. California Spray-Chemical Corp. Maryland Heights, Miss.
Chemagro B-1843 -- Experimental sample, composition unknown. Chemagro Corp. New York, N. Y.
Bayer 22555 (Dexon) -- *p*-Dimethylaminobenzenediazo sodium sulfonate. Chemagro Corp. Kansas City, Miss.

Onion seed of the variety Ebenezer was treated by mixing equal weights of fungicide and seed in a 250 ml flask. All but 5 of the treatments were applied to seed previously wetted with a 2% methyl cellulose (400 centipoise) solution. To control onion maggots in the field experiment, 50% heptachlor (2.25 mg per 9 g of seed) was mixed with the fungicide and seed in all treatments which were not fungicide-insecticide combinations. After thorough mixing in the flask the contents were emptied into a sieve to remove excess chemical not adhering to the seed. The total weight of fungicide and seed

was determined to calculate the rate of application. This varied between products as some adhered to the seed more readily than others.

Field and greenhouse experiments were carried out with the various seed-treatment fungicides. The land used for the field experiment had produced the previous year a crop of onion sets which were severely infected with smut. Three grams of seed of each of 18 treatments were sown in 6-ft. rows randomized in each of 3 replicates. In the greenhouse experiment 20 treatments randomized in each of 4 replicates were compared in soil artificially infested with a spore suspension of U. cepulae. The seed was planted at a rate of 1.5 g per 44-in. row. The onions were harvested, washed, and examined for smut 2 months after planting.

Results

The results of these tests are shown in Table 1. F values obtained from analyses of variance were not significant for replicates but were significant at the 1% level for stand and smut percentages (transformed to degrees) in both tests. No damage from onion maggot was evident and the significant stand reductions were attributed to phytotoxicity of the pesticide.

Formulations containing 80% hexachlorobenzene successfully controlled smut and increased stand. Although some of the products containing 40% hexachlorobenzene gave good control of smut, considering the lower rate of application, further tests should be made because Duran and Fischer (2) found variations in phytotoxicity between lots of the same brand manufactured in different years. Two of the products containing hexachlorobenzene, Dual Purpose Bunt-no-more and Sanocide, were phytotoxic.

Captan and products containing thiram, when pelleted with the seed, were also satisfactory. Of the latter group, only the experimental sample of Heptachlor-Thiram was phytotoxic as indicated by the significant reduction in stand. Captan applied to dry seed effectively controlled smut for the reason suggested by Larson and Walker (3) that the amount of fungicide applied with seed sown at a heavy rate for set production approximates that applied in the furrow by Newhall et al (5) for smut control. The percentage of smut in the best treatments in the greenhouse appears high but most of the plants rated smutty in these treatments were only lightly infected and would probably recover, as reported by Duran and Fischer (2). Counts were made on 2 replicates of each treatment in the greenhouse and the percentages of severely smutted plants ranged from 2 to 7% in the rows planted with seed pelleted with Captan, Arasan, and 80% hexachlorobenzene.

Chemagro B-1843 and Bayer 22555 (Dexon) are not recommended for smut control but were included on a trial basis. Each was phytotoxic when pelleted with onion seed.

Table 1. The Effect of Seed Treatment Fungicides on Smut Infection and Stand in Onions Sown for Set Production

Treatment	Field			Greenhouse			Ratio of fungicide to seed
	Stand	Smut		Stand	Smut		
		Per cent	Degrees 1/		Per cent	Degrees	
Check	412	13.1	21.2				
Talc (P) 2/	368	23.5	28.8	301	83.1	65.8	
Heptachlor (P)	279	11.2	19.4	301	86.3	68.5	
Captan 50-W	520	2.6	8.8				
Captan 50-W (P)	326	3.7	9.8	354	31.1	33.8	8:100
Arasan 75	523	7.4	15.6	353	26.5	31.0	65:100
Arasan 75 (P)	317	3.6	10.6	302	50.5	45.3	8:100
Delsan A-D (P)				335	20.7	27.0	62:100
Panoram D-31 (P)				301	25.3	29.3	41:100
Heptachlor-Thiram (P)	105	2.3	8.6	338	26.8	31.0	45:100
Anticarie 80 (Ros) (P)	335	2.0	6.6	358	15.1	23.0	58:100
Anticarie 80 (FD) (P)	293	2.6	9.0	354	28.9	32.8	76:100
Ortho 80 (P)	333	3.6	10.5	352	27.8	31.8	18:100
HCB 100 (P)	284	6.4	14.5				
Bunt-no-more (P)	421	8.3	16.0	351	29.0	32.5	23:100
No-bunt (P)	250	4.6	12.1	321	38.5	38.0	18:100
Co-op Hexa (P)	433	6.2	14.0	321	32.7	35.0	23:100
Anticarie 40 (P)	330	3.8	10.6	339	40.2	39.3	16:100
Shell Aldrin-HCB (P)				219	43.1	41.0	6:100
Dual Purpose Bunt-no-more (P)	46						
Sanocide (P)	8						
Chemagro B-1843				299	42.1	40.5	25:100
Chemagro B-1843 (P)				151	37.2	37.5	46:100
Bayer 22555				200	54.0	47.5	0.2:100
Bayer 22555 (P)				1			13:100
L.S.D. (.01)	165		9.1	50		12.3	

1/ Degrees obtained from angular transformations of smut percentages

2/ (P) = pelleted

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EPIDEMIOLOGY OF FUSARIUM STORAGE ROT OF POTATOES IN PRINCE EDWARD ISLAND[1]

G. W. Ayers[2] and G. C. Ramsay[3]

Potato storage rot, or dry rot, caused by Fusarium sambucinum Fckl. f. 6. Wr. reached epidemic proportions in Prince Edward Island in 1960. An intensive study of the disease has shown that the high level of infection was influenced by (1) the susceptibility of the variety Sebago which was widely planted (2) the amount of inoculum carried on the seed-piece (3) the degree of soil infestation by the pathogen at time of harvest as influenced by weather conditions during the summer and (4) the amount of injury to the tubers during harvesting and grading.

Economic losses due to Fusarium rot were not encountered in Prince Edward Island prior to the introduction of the highly susceptible variety Sebago, and although the disease has been observed each year, it was only in 1946, 1947 and 1960 that dry rot posed a serious problem to growers and shippers.

Published data (1) on experiments conducted at Charlottetown show that F. sambucinum enters the soil at time of planting in the form of spores adhering to the surface of the seed-piece and that effective control may be obtained through seed-piece treatment with an organic mercury fungicide. The extent of propagation of the pathogen in the soil is apparently dependent on the weather conditions that prevail during the growing season. Meteorological data for the crop production seasons in the years 1945-1960, together with information on the extent of Fusarium decay, are presented in Table 1.

It has been observed that epidemics of storage rot follow growing seasons in which conditions are characterized by low soil moisture, high rates of evaporation and above-average air temperatures and hours of sunshine. Rainfall for the period July - September was very light in 1945 and 1946, the evaporation rate was much higher than normal, and storage rot was prevalent in harvested crops. Heavy losses in the 1947 crop can not be correlated with low rainfall but a high evaporation rate together with above-normal temperatures and hours of sunshine during the growing season brought about drought conditions in many localities in Prince Edward Island. The sharp decline in the extent of decay found in the 1948 crop was concomitant with high soil moisture during the growing season. Rainfall was adequate, the evaporation rate was low and hours of sunshine were considerably below those in epidemic years.

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Table 1. The occurrence of Fusarium storage rot in Prince Edward Island under varying weather conditions in the period 1945-1960.

	Average meteorological data recorded at Charlottetown, June - September				
	Rainfall (inches)	Evaporation rate (inches)	Air temperature °F	Sunshine (hours)	Estimated losses from storage rot
1945	2.48	4.65	63.0	221.7	moderate
1946	2.71	4.42	62.9	229.3	heavy
1947	3.45	4.19	63.6	234.7	heavy
1948	3.39	3.20	62.2	214.9	light
1949-58	3.19	3.55	61.9	213.0	light
1959	2.80	3.05	62.7	178.4	light
1960	2.34	4.00	64.1	239.4	heavy

The period 1949-58 was characterized, for the most part, by excellent tuber yields and a combination of meteorological conditions which were not favorable for the disease. The epidemic in 1960 was preceeded in 1959 by a crop showing very little Fusarium decay. Although the 1959 rainfall was below average, evaporation was limited and soil moisture levels proved adequate for plant growth.

Losses due to Fusarium decay in 1960 were greater than in any previous year and it may be observed (Table 1) that meteorological conditions favored dessication of the soil to an extent not exceeded in any other period covered in this survey. During 1960, drought was more severe and storage rot more prevalent in Prince than in Queens and Kings counties. The data presented in Table 2 were compiled by the Seed Potato Inspection Service.

Table 2. The occurrence of Fusarium storage rot of potatoes in farm storages in the three countries of Prince Edward Island in 1960

County	Farm storages surveyed	Average per cent rot
Prince	248	8.15
Queens	84	2.50
Kings	37	1.82

Further data on potato yields and the extent of decay were assembled from areas where rainfall was recorded. The results of this survey are presented in Table 3.

Table 3. The occurrence of Fusarium storage rot of potatoes in certain localities in Prince Edward Island.

District	County	Average monthly rainfall (June-Sept. (inches)	Yields in bushels 1960	Yields in bushels (average)	Per cent rot
O'Leary	Prince	1.36	225	352(13 yrs.)	8.4
Urbanville	Prince	2.40	300	328(15 yrs.)	1.5
New London	Queens	2.50	250	284(19 yrs.)	2.1
Charlottetown	Queens	2.34	287	287(3 yrs.)	2.5
Alliston[1]	Kings	3.28	143	177(13 yrs.)	0.5
Monticello	Kings	2.43	350	251(19 yrs.)	1.3

[1] Very light soil.

A much greater extent of decay occurred in the O'Leary district than in other districts in Prince Edward Island, and the severity of the epidemic there can be correlated with very low rainfall. The Seed Potato Inspection staff has further noted that the pockets of heavy infection that occurred in other sections of the province in 1960 were associated with severe drought. Fusarium rot was not prevalent in Kings County and the limited infection was associated with adequate soil moisture levels during the critical period of tuber growth.

In assessing the factors associated with the occurrence of dry rot, consideration must be given to soil moisture levels at time of harvest. The pathogen enters the tubers through wounds contracted at harvest, and tuber injury from contact with digger chains and bars is more likely to occur when the soil is very dry.

A substantial portion of the Prince Edward Island potato crop is harvested with the elevator-type digger and observations made in 1960 indicate that dry rot became readily established in bruises that occurred as the result of the passage of tubers over the bars of this type of machine. It was generally noted that tuber abrasions were less numerous and subsequent decay less where the crop was harvested with the beater and digger-picker types of harvesters. In harvesting with the beater digger the tubers come less in contact with metal machinery parts then with the elevator digger, and in digger-picker harvesting the crop must of necessity be handled slowly. Injury similar to that encountered with the elevator digger can occur when the crop is removed by combine harvesters. An essential precaution in potato lifting with elevator and combine harvesters is to keep tractor speeds at a minimum.

A controlled inoculation experiment conducted in the winter of 1961 showed no differences in the susceptibility to storage rot of Sebago tubers produced under dry soil conditions and those produced under conditions of adequate moisture.

In summarizing the occurrence of storage rot in the 1960 potato crop in Prince Edward Island it is concluded that the greatest single factor contributing to the severity of the disease was the relatively high extent of soil infestation by the pathogen and that the propagation of the organism was favored by dry, warm soil during the period of plant growth.

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INTRODUCTION OF A VIRUS TO MCINTOSH APPLE FROM AN IMPORTED CLONE OF GRANNY SMITH[1]

Maurice F. Welsh[2] and F. W. L. Keane[3]

There have been several reports of the introduction, or suspected introduction, of viruses in fruit trees imported to one country or continent from another. Milbrath (5) has provided a well documented report of the presence in Esperen cherry imported to the United States from France of a virus causing symptoms of Pfeffingerkrankheit in Bing cherry. Brase and Gilmer (3) have recorded the presence of rubbery wood virus in a clone of Malling I apple rootstock originally imported from England. The work of Reeves, Cheney and Milbrath (6), and Wilks (9), has provided evidence for introduction of little cherry virus in imported flowering cherry trees.

This is a report of the demonstration of a potentially serious virus occurring without symptoms in Granny Smith apple trees that were imported to British Columbia from New Zealand.

Experimental Evidence

The indicator hosts that are used in other regions have been included in host range studies of British Columbia apple viruses, in the expectation that some of the virus diseases being demonstrated in British Columbia apple plantings are manifestations of diseases described in other varieties elsewhere.

Granny Smith is the variety in which the symptoms of green crinkle and apple ring spot have been described in greatest detail by New Zealand workers (1,2). Trees of Granny Smith had been imported from New Zealand by a British Columbia grower in 1939. The trees had been purchased from a New Zealand nursery, and planted in a small block at East Kelowna, B. C. In 1958, when scions of Granny Smith were required for the host range studies at Summerland, 3 trees of the original planting remained. Two of these trees displayed mild symptoms of apple mosaic. The third tree, examined carefully in 1958, 1959, and 1960, has displayed no foliage symptoms, and was selected as a scion source. Each season, several scattered fruits were found on this and the neighbouring Granny Smith trees that bore minute areas of russeting. However this low proportion of mildly russeted fruits was not deemed justification for considering the trees to be diseased.

The first test of Granny Smith was designed to determine its reactions to the leaf pucker disease (7,8). Scions of this variety were topworked in 1958 to 2 limbs each of 3 trees in test plots. These trees were part of a block of 19 bearing McIntosh trees that had been used in leaf pucker transmission tests.

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Ten of the trees were of a striped strain of McIntosh that has now proved to be almost completely tolerant to leaf pucker. The remaining 9 trees are of a blushed strain that displays foliage and fruit symptoms when inoculated with leaf pucker. Two of the trees to which Granny Smith was topworked (Trees 1 and 2), were of the tolerant striped strain, inoculated with leaf pucker in 1955. Tree 3 was of the sensitive blushed strain, but had served as an uninoculated check in the leaf pucker tests. Trees 1 and 2 have displayed mild foliage symptoms of leaf pucker in several seasons since their inoculation in 1955. They began to crop in 1957, bore heavy crops in 1958, and displayed no fruit symptoms in either of the se seasons prior to the topworking of Granny Smith on them. The uninoculated Tree 3 displayed neither foliage nor fruit symptoms prior to the topworking of Granny Smith on it.

In 1959, the year following application of Granny Smith scions to the 3 trees, they bore a light crop. One of the 3 McIntosh fruits on Tree 1 was recorded as having shallow skin depressions. Two of the 33 McIntosh fruits on Tree 3 had similar symptoms. There were no symptoms on the fruits of Tree 2.

In 1960, all 3 trees bore McIntosh fruits with symptoms that had not been seen previously in British Columbia. Patches of coarse scurfy russeting were evident by mid-June. By early September these russeted areas lay within large depressions on the cheeks of the fruit (Fig. 1A). The depressions were underlaid by light green watersoaked flesh, extending to the core (Fig. 1B). The numbers of affected fruits were as follows: Tree 1 - 103/131; Tree 2 - 56/111; Tree 3 - 14/147. All of these fruits were so severely affected that they would be downgraded or culled. There were additional fruits on all 3 trees that were misshapen but not russeted. No foliage symptoms were observed. All other trees of the sensitive McIntosh strain bore fruits with symptoms characteristic of the leaf pucker syndrome (Fig. 2). The other trees of the tolerant McIntosh strain bore normal fruits, or a small proportion of fruits with mild symptoms of the leaf pucker syndrome.

Two of the Granny Smith fruits borne in 1960 on Tree 2, and the one fruit borne on Tree 3, were slightly misshapen, but displayed no russeting symptoms. There were no symptoms on the leaves of Granny Smith branches in 1959 and 1960.

Discussion

There appears to be adequate evidence that a virus was introduced from the Granny Smith scions to the McIntosh trees. The external symptoms induced on McIntosh fruits were quite dissimilar to the much less obvious skin depressions, and the rings of very fine russeting, that occur on the fruits of trees affected by leaf pucker (Fig. 2). There were symptoms in flesh tissues, whereas fruits borne on trees with leaf pucker have never displayed symptoms in the flesh. Moreover the symptoms occurred on 2 trees of a McIntosh strain that has proved almost completely tolerant to the leaf pucker virus, and on a tree that had not been inoculated with leaf pucker.

The assumption seems justified that this virus was present in the Granny Smith tree when it was imported from New Zealand, because the fruit

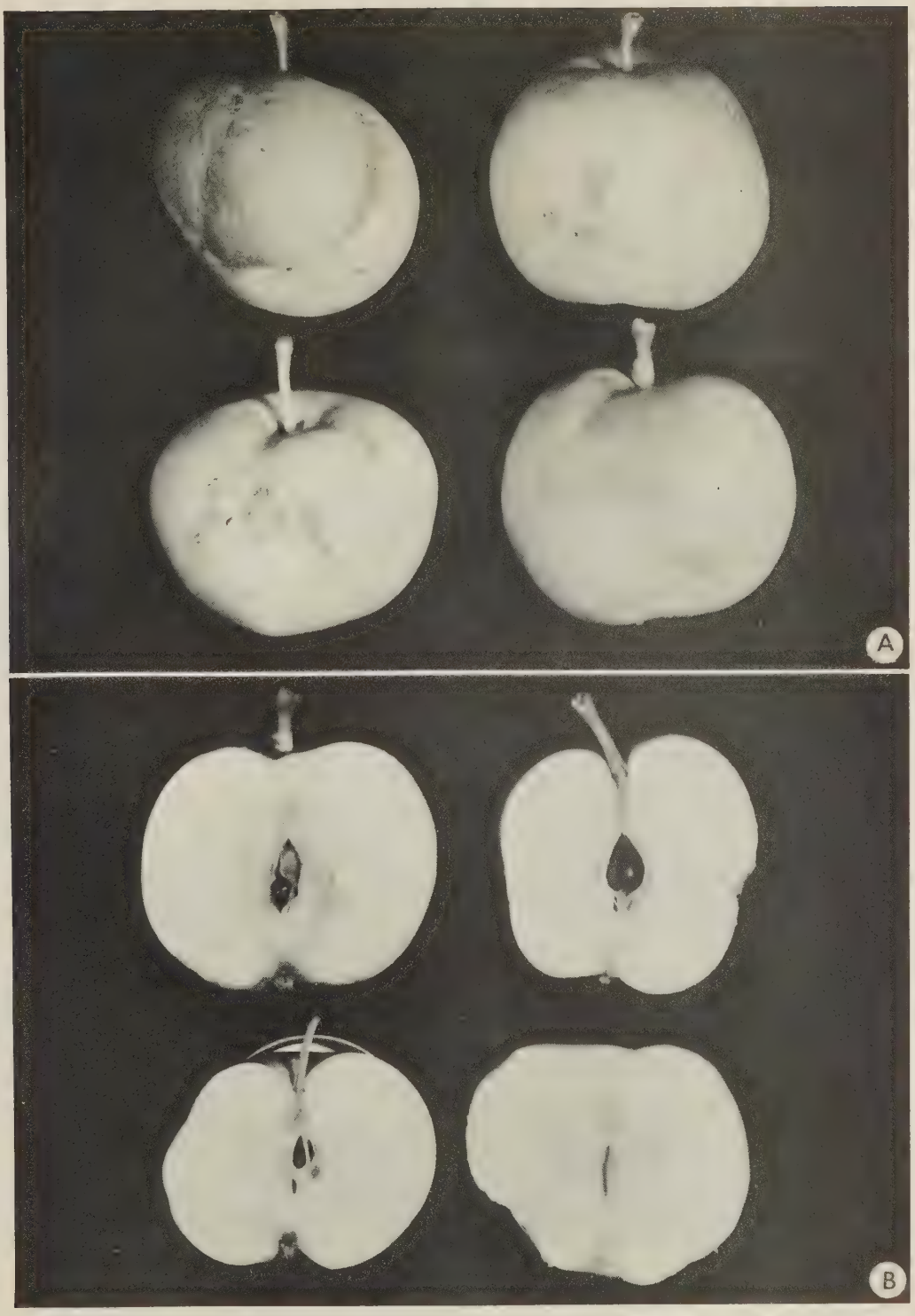

Figure 1. McIntosh fruits borne on tree topworked to Granny Smith. A) Coarse scurfy russeting on depressed areas of fruit B) Watersoaked flesh extending from russeted depressions to the core.

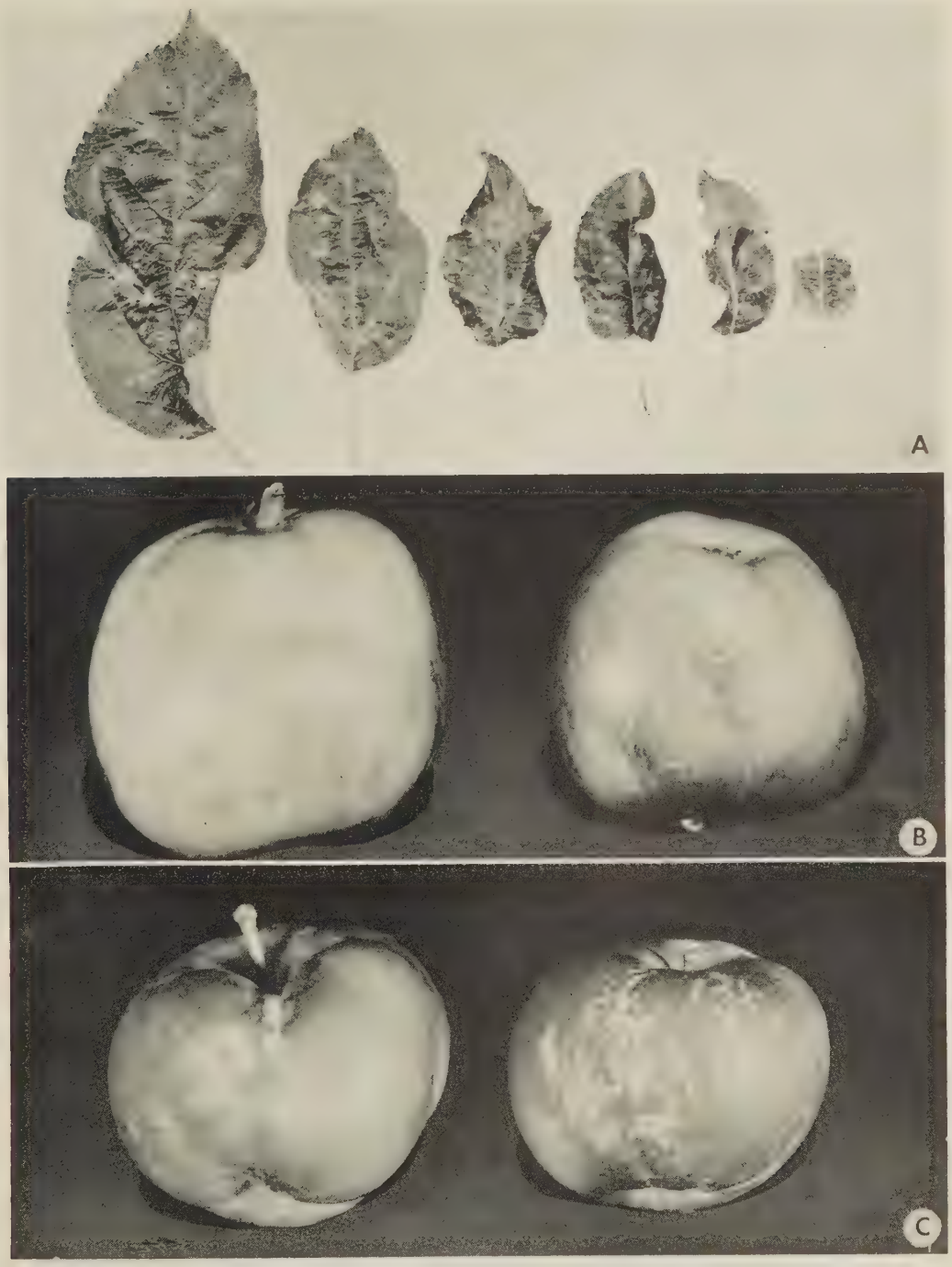

Figure 2. Leaf pucker of McIntosh. *A)* Leaf symptoms *B)* Pitting symptoms *C)* Russet symptoms.

symptoms that were induced have not been observed elsewhere in British Columbia, or reported from other parts of western North America. As McIntosh has not been grown extensively in New Zealand, or included in host range studies of New Zealand virus diseases, it is not possible so far to identify this as one of the viruses responsible for diseases occurring in New Zealand. Fortunately, the virus does not appear to spread naturally in British Columbia. The imported Granny Smith trees have been growing beside a block of McIntosh trees since 1939, without appearance of symptoms in the McIntosh.

There is a probability that the apple mosaic observed in 2 of the Granny Smith trees at East Kelowna was also in the trees when they were imported. British Columbia records of apple mosaic include only 3 other trees in 2 widely separated orchards, whereas this disease is common in New Zealand plantings (4).

This report adds evidence that there is danger, as emphasized by Milbrath (5), of innocently introducing plant viruses in symptomless species and varieties. However, it is not considered justification for exclusionary measures. Rather, it accentuates the need for thorough indexing of propagating materials used by nurseries in producing trees either for domestic use or for export. In recent years New Zealand has been a leader in the development of tree fruit nursery certification measures. Although this virus introduction was possible in 1939, it is highly improbable that it could occur in nursery stock imported from New Zealand in 1960.

The British Columbia grower has been advised to remove his imported Granny Smith trees, and all infected materials in test plots have been destroyed.

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AN ASSESSMENT OF APPLE VIRUS INDEXING RESULTS[1]

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Abstract

Assessments have been made of the results of indexing apple trees in British Columbia plantings, and of observations in virus-diseased orchards. The viruses responsible for stem pitting of Virginia crab, decline of Virginia crab, and rubbery wood in Lord Lambourne can occur independently. The virus responsible for flute fruit of Virginia crab can occur in the absence of those causing rubbery wood, and decline of Virginia crab. Prunus tomentosa, Russain apple seedling R12740-7A, Hopa crab, and Bedford crab have displayed symptoms when topworked on Malling II rootstocks although Lord Lambourne and Virginia crab topworked on the same rootstocks display no symptoms. Stem pitting and dwarf fruit can occur separately on Hyslop crab, and either can occur on trees that do not show decline symptoms. Virginia crab has displayed no symptoms in the season following inoculation from Hyslop crab displaying dwarf fruit, stem pitting, and decline symptoms. These results suggest need for caution in interpreting results from indexing on a limited range of indicators.

Introduction

During the first few years in which viruses are investigated in a plant genus, there is an expectation that a number of indicator hosts will be found, and that confusion will surround the identities and relationships of the viruses to which these indicator hosts are sensitive.

Usually successions of cross transmissions among carefully selected and systematically employed indicator hosts provide gradual clarification. However the planning of such an indexing program must depend on the assessment of data arising incidentally from earlier investigations of known diseases.

Useful information has been assembled during the investigation, initiated in 1955, of viruses occurring in British Columbia apple plantings. A major part of the original project was intended to determine the prevalence of stem pitting and rubbery wood viruses in orchard trees and scion source trees. The indicator hosts used most consistently have been Virginia crab and Lord Lambourne. Results of this indexing to 1958 (15) demonstrated that at least four viruses can occur as latent infections in the trees of British Columbia apple plantings. The 1958 report can now be augmented by additional readings in the indexing of the listed source trees, and results from the indexing of additional trees.

Selected source trees, for which indexing on Virginia crab and Lord Lambourne have indicated differences in virus content, have been indexed

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also seedlings of Prunus tomentosa Thunb.

Since 1958 the occurrence of other viruses has been demonstrated, in orchard surveys, and in the course of attempts to establish test trees of additional indicator hosts when Malling II was used as a standard rootstock. The varying behaviour of some of the indicator varieties when topworked on the supposedly virus-free Malling II has revealed the presence of a virus, or viruses, to which a number of these varieties are sensitive.

This paper presents data derived from these observations, and an assessment of their significance in cataloguing viruses that can occur as latent infections in apple.

Materials and Methods

The materials and methods used for all indexing on Virginia crab, Lord Lambourne and P. tomentosa were those described in an earlier report (15). All test trees were grown on Malling II rootstocks derived from indexed stoolbeds at East Malling Research Station. All test trees were paired in orchard plots with an uninoculated test tree, the latter growing between the two inoculated trees. For all reported results the two test trees gave identical readings, and the check tree remained healthy.

The additional indicator hosts that have been assembled are Hopa crab, Bedford crab, Columbia crab, Transcendent crab, Hyslop crab, and the Russian apple seedling R12740-7A introduced as an apple virus indicator by Mink and Shay (11). All have been grafted to Malling II rootstocks derived from the same stoolbeds that provided rootstocks for the Virginia crab and Lord Lambourne test trees. Indexing at East Malling had shown Malling II to be free from all viruses of apple recognized before 1958. The rootstocks were grafted to variety while growing in one-gallon cans in the greenhouse, and have been retained in the greenhouse during the full period that they have been under observation.

Results and Discussion

The results of indexing on Virginia crab, Lord Lambourne and P. tomentosa from 1955 to 1960 are summarized in Table 1.

The reactions of various Malus species and varieties, when attempts were made to propagate them on Malling II rootstocks, are described in Table 2.

Orchard observations on a block of Hyslop crab trees are reported in Table 3.

An evaluation of the results recorded in Tables 1, 2 and 3 suggests that there are numerous viruses, or strains of viruses, occurring commonly in apple, and characteristically latent in commercial apple varieties. The manifestations of virus action that have been recorded at Summerland and elsewhere in these test plants include: stem pitting of Virginia crab (6, 7, 15, 17); decline of Virginia crab (15, 17); flute fruit of Virginia crab (17); stem pitting of Hyslop crab (2, 4, 7); decline of Hyslop crab (2, 15, 17); dwarf fruit of Hyslop crab (2, 3, 5, 17); rubbery wood of Lord Lambourne and other apple varieties (9, 15, 17); leaf flecking of Hopa crab (14, 17); stem pitting of Hopa

crab (1); leaf necrosis of Bedford crab (17); chlorotic leaf spot of Russian apple seedling R12740-7A (11); and foliage mottling of P. tomentosa (10,15).

There is an inclination to assume that these disease manifestations can be grouped, and each group attributed to the action of one virus. This leads to the assumption that a selected indicator can serve to indicate freedom from the viruses to which the other hosts are sensitive. An examination of the data recorded in Tables 1, 2 and 3 suggests that considerable caution must be exercised in attributing to single viruses the responsibility for causing more than one of the reactions that have been observed in the range of apple virus indicators. Most of the virus manifestations in the indicators used have been induced by one or more sampled apple sources that failed to induce the other virus manifestations.

Stem pitting of Virginia crab and rubbery wood of Lord Lambourne.

When indexing was initiated at Summerland, there existed a possibility that stem pitting was a manifestation of the rubbery wood virus in Virginia crab. The indexing results recorded in Table 1 and elsewhere (13,15) have shown that rubbery wood virus and stem pitting virus can occur independently.

Stem pitting of Virginia crab and decline of Virginia crab.

Decline of Virginia crab has occurred, so far, only after inoculation with sources carrying both stem pitting and rubbery wood (Table 1). However, decline was not induced by sources 9D-90910, Evans 1, Skelly H-20 and Splett 1, which carried both stem pitting and rubbery wood, by GG-13 which carries rubbery wood but not stem pitting, and by 6 sources that carry stem pitting but not rubbery wood. This justifies the conclusion that decline is not caused by the stem pitting virus or rubbery wood virus alone, or by combined action of the two viruses.

Stem pitting of Virginia crab and chlorotic leaf spot.

Russian apple seedling R12740-7A has displayed chlorotic leaf spot when propagated on Malling II rootstocks (Table 2). Over 200 Virginia crab trees have been propagated on these stocks, and none has shown stem pitting unless it was used to index diseased apple clones. Source 9D-90514 (Table 1), that has induced stem pitting in Virginia crab, has not caused chlorotic leaf spot symptoms in Russian apple seedling R12740-7A in 2 seasons following inoculation. Thus, although many apple sources induce both stem pitting and chlorotic leaf spot, it appears possible for the viruses responsible for these reactions to occur independently. Cation and Carlson (4) have reported a similar demonstration of independent occurrence of these viruses. The stem pitting symptom on Russian apple seedling R12740-7A recorded by other workers (11) has not been found on this host when propagated on Malling II.

Stem pitting of Virginia crab and the reactions of Hopa and Bedford crabs.

Hopa and Bedford crabs have developed foliage flecking and necrosis, and tip dieback symptoms, when propagated on Malling II rootstocks (Table 2). As Virginia crab is symptomless when grown on this rootstock, the stem pitting of Virginia crab is apparently not caused by the virus responsible for the foliage symptoms on Hopa and Bedford crab. Stem pitting symptoms (1)

have not been observed on Hopa and Bedford crabs propagated on Malling II. Tests to compare the reactions of Malus platycarpa Rehd., described by Luckwill and Campbell (8) with those of Hopa, Bedford, and Virginia crab are incomplete.

Decline of Virginia crab and decline of Hyslop crab.

The symptoms of decline in Virginia crab and in Hyslop crab are similar, and both have been invariably accompanied by stem pitting symptoms (2,15). However, Virginia crab test trees that in 1959 received buds from sources Walburn B-69 and Walburn B-71 (Table 1), both of which are Hyslop trees in advanced state of decline, have shown no evidence of decline in 1960. As Virginia crab test trees characteristically show severe decline in the season following inoculation with Virginia crab decline virus, this test suggests that the virus causing decline of Hyslop crab does not induce decline in Virginia crab.

Stem pitting of Hyslop crab and decline of Hyslop crab.

The trees B-25, B-31, B-43, B-63, B-69 and B-71 (Table 3) are part of a commercial Hyslop planting. Trees B-69 and B-71, which have been topworked to Jonathan, show severe symptoms of both stem pitting and decline. Trees B-25, B-31, B-43 and B-63, have not been topworked. Tree B-43 displays severe stem pitting, and trees B-25, B-31 and B-63 display moderate stem pitting, but these trees show no evidence of decline. Other trees in the block and in other surveyed blocks display mild stem pitting, but no decline. Thus it is possible for stem pitting to occur on Hyslop crab with or without the onset of decline.

Flute fruit of Virginia crab, and stem pitting and decline of Virginia crab.

Flute fruit characteristically occurs on Virginia crab trees that display stem pitting symptoms (Table 1). So far there have been no exceptions to the co-occurrence of these two types of symptoms on this host. Flute fruit and decline also appear to be distinct. All trees affected by decline that have fruited have displayed flute fruit symptoms. However flute fruit has been observed on many orchard and test trees not affected by decline.

Dwarf fruit of Hyslop crab, and stem pitting and decline of Hyslop crab.

Dwarf fruit characteristically occurs on Hyslop crab trees that display stem pitting. However, their co-occurrence has not been invariable (Table 3). In the Walburn orchard, trees B-31 and B-63, which display moderate stem pitting, have borne normal fruits in 2 successive seasons. Trees B-15 and B-31, which are free from stem pitting, have borne fruits with mild dwarf fruit symptoms in these two seasons. Surveys of a second orchard have provided a similar example of general but not invariable co-occurrence. Dwarf fruit and decline also appear to be distinct. Trees B-69 and B-71, suffering severe decline, have displayed severe dwarf fruit symptoms. However fruits with mild to severe symptoms have been borne by trees that do not show decline, in the two surveyed orchards.

Table 1. Results of the indexing of apple source trees on Virginia crab, Lord Lambourne apple, and Prunus tomentosa.

Source	Flute fruit	Virginia crab stem pitting	Rubbery wood	Virginia crab decline	P. tomentosa mottle
E.F. 9J-P30	-	-	-	-	-
Malling II	-	-	-	-	+
E.F. 9D-91708	-	-	-	-	no index
Hait BB-22	-	-	-*	-	+
Hait U-17	+	+	-	-	no index
E.F. 9D-91912	+	+	-	-	no index
Hait V-12	+	+	-	-	no index
E.F. 9C-7349	+	+	-	-	no index
E.F. 9D-90514	+	+	-	-	no index
Q-7	+	+	-	-	no index
Walburn B-69	?	-*	-*	-*	no index
Walburn B-71	?	-*	-*	-*	no index
Walburn B-43	?	-*	-*	-*	no index
Hait GG-13	-	-	+	-	+
Evans 1	+	+	+	-	+
Splett 1	+	?	+	-	+
Skelly H-20	+	+	+	-	no index
E.F. 9D-90910	+	+	+	-	+
Q-13	?	+	+	+	no index
WP-1	+	+	+	+	no index
E.F. 3-17-7	+	+	+	+	+

? Indexing incomplete
* Readings for one season only

Table 2. Symptoms displayed by apple virus indicator hosts when propagated on E. M. II rootstocks.

Russian Seedling R12740-7A	- High percentage scions remained dormant, eventually died. Dwarfed shoots. Chlorotic leaf spot.
Bedford crab	- High percentage scions remained dormant, eventually died. Dwarfed shoots. Leaf necrosis, defoliation. Tip dieback.
Hopa crab	- High percentage scions remained dormant, eventually died. Dwarfed shoots. Purple leaf flecking.
Virginia crab	- No symptoms. Vigorous growth.
Hyslop crab	- No symptoms. Vigorous growth.
Transcendent crab	- No symptoms. Vigorous growth.
Columbia crab	- All scions remained dormant, eventually died.
Prunus tomentosa	- Foliage mottling.

Table 3. Occurrence of stem pitting, dwarf fruit, and decline of Hyslop crab in Walburn orchard, Kelowna, B.C.

	Stem pitting	Dwarf fruit	Decline
B15	none	mild	none
B17	none	none	none
B19	none	none	none
B21	trace	none	none
B23	trace	mild-moderate	none
B25	moderate	mild-moderate	none
B31	moderate	none	none
B33	none	mild	none
B35	mild	none	none
B37	mild	none	none
B41	mild	none	none
B43	severe	moderate-severe	none
B47	mild	mild-moderate	none
B63	moderate	none	none
B67	mild	mild-moderate	none
B69*	severe	moderate-severe	severe
B71*	severe	severe	severe

* Topworked to Jonathan.

Significance of P. tomentosa leaf mottling.

Transmission results (Table 1) have demonstrated that leaf mottle of P. tomentosa can be induced by apple sources free from the viruses that cause stem pitting (3 trees), flute fruit (3 trees), rubbery wood (2 trees) and Virginia crab decline (6 trees). Moreover, P. tomentosa has displayed foliage mottling when used to index Malling II, which has not induced decline of Hyslop crab. So far, results have not been obtained from tests in which P. tomentosa has been used for the indexing of materials known to be free from chlorotic leaf spot, or the viruses that cause symptoms in such crab varieties as Hopa and Bedford. At present it seems safe to conclude that P. tomentosa is not a differential indicator for the viruses causing stem pitting, decline, or flute fruit of Virginia crab, or for those causing decline of Hyslop crab, or rubbery wood. However, its status as an indicator for chlorotic leaf spot and the leaf flecking diseases of species and varieties of crabapple, has not been demonstrated.

Conclusions

The results of transmission tests at Summerland and elsewhere have demonstrated that viruses are prevalent in apple plantings, and that a number of viruses or virus strains are involved. Their designation either as viruses or virus strains is considered academic until information is available on their interactions in their hosts, and their chemical, physical, and serological characteristics. At present the significance of their differences is in their impact on the assessment of indexing results.

Obviously the results that have been recorded are not sufficiently comprehensive to justify assumption of unique etiology for all of the disease manifestations that are described and assessed. They do provide justification for extreme caution in assuming that the symptoms displayed by any one indicator host used in apple indexing are attributable to the viruses responsible for either the same or different symptoms in other indicators. They suggest equal caution in categorical assumption of virus freedom on the basis of indexing on a limited range of indicators. They emphasize the need for expanded indexing of apple trees and rootstock clones in an effort to elucidate the relationships of the viruses to which the indicator hosts are sensitive. Such tests are now in progress at Summerland, with a wider range of test plants employed for indexing of all apple sources that have differed significantly in their effects on one or more of the indicator hosts used so far.

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STRAWBERRY VIRUSES[1]

Frances C. Mellor[2] and Randal E. Fitzpatrick[3]

Introduction

The majority of strawberry viruses do not produce, on commercial varieties, symptoms that are sufficiently characteristic to allow positive identification in the field. Transmission to indicator plants is necessary for the detection and identification of these viruses.

When, as often happens, more than one virus is present, the separation of the components of the mixture can sometimes be achieved by making use of differences in their vector relationships or differences in their stability when the plants are grown at high temperatures.

Before entering upon a detailed description of the various viruses, it will be useful to make some general comments on the indicators and techniques we have used in our studies in British Columbia.

INDICATOR PLANTS

Several strains of the wild strawberry, Fragaria vesca L., are used as indicators. No one of these strains is adequate for the detection of all the known strawberry viruses but, by using discrimination, a minimum number of indicators may be selected for a complete indexing program.

East Malling clone of Fragaria vesca (E M C)

This clone was originally selected at East Malling, England, because of its sensitivity to mottle viruses. This sensitivity is now known to be due to the presence of latent-A virus with which the clone is infected. The East Malling clone is a good indicator for mottle viruses, for veinbanding, and for latent-C, but it is less sensitive to mild yellow-edge than some of the other indicators. It is a poor indicator for crinkle because latent-A is apparently a strain of, and affords partial protection against, the crinkle virus.

Alpine Fragaria vesca seedlings

The Alpine Fragaria vesca is a runnerless strain grown from seed. The seedlings are not entirely uniform in their reaction to any given virus isolate. They are satisfactory indicators for mild yellow-edge and for severe strains of mottle or veinbanding, but are poor indicators for mild strains of mottle and veinbanding. They are not sensitive to latent-C virus.

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1 Contribution No. 31 from the Canada Agriculture Research Station, Vancouver, B.C.

2, 3. Plant Pathologist and Director, respectively.

Frazier's runnering alpine seedling (U C 1)

This is a runnering strain of F. vesca, selected by N.W. Frazier of Berkeley, California. This clone was developed from an Alpine seedling and it is assumed to be a hybrid of Alpine and East Malling F. vesca. It is a good indicator for both crinkle and mild yellow edge. It is not sensitive to latent-C, and is less sensitive to mottle and veinbanding than is the East Malling clone of F. vesca.

Miller's virus-free Fragaria vesca

This clone was selected by Paul W. Miller at Oregon State College. It probably arose from a seedling of the East Malling F. vesca. It is free of latent-A virus, and symptoms on this indicator are similar to those on Frazier's U C 1 clone.

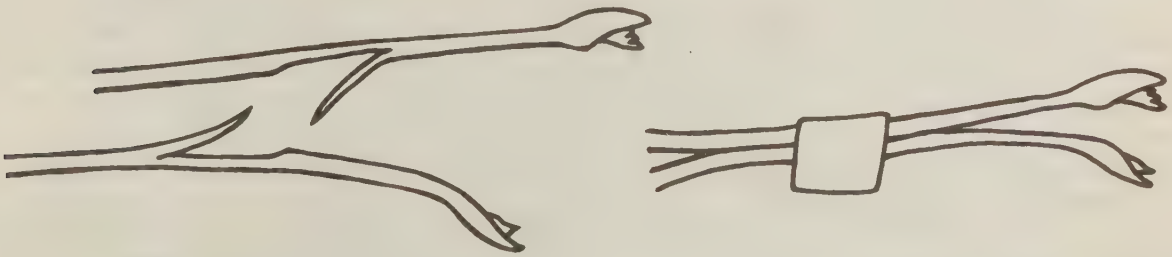
Fulton's latent-free Fragaria vesca (E M K)

This is a clone developed by J.P. Fulton of Arkansas, from a plant of the East Malling clone which he had freed of latent-A by heat treatment. Symptoms of mild yellow edge and latent-C on this indicator are similar to those on the East Malling clone. Symptoms of mottle and veinbanding are similar to those on Frazier's U C 1 and Miller's clone.

TECHNIQUES

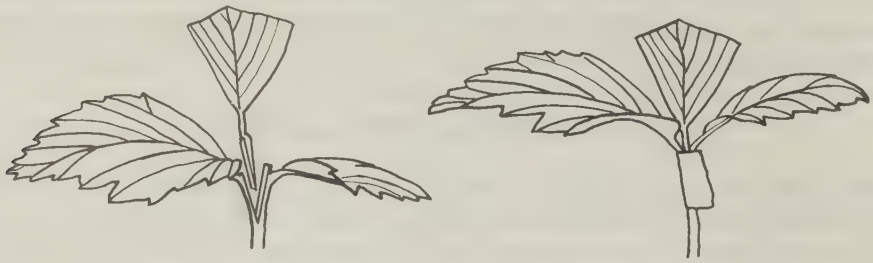
Grafting

Runner grafting, described by Harris and King (14), has been widely used for the experimental transmission of strawberry viruses. If this technique is used in indexing commercial stock, a non-grafted sister plant should be used for propagation because of the possibility of transmission of a latent virus from an infected indicator.



In runner grafting, young stolons from the donor and indicator plants are grafted together as shown above. A slanting cut, about three quarters of an inch long, is made in each of the stolons. The cut is made toward the stolon tip of the donor plant, and toward the stolon base of the indicator plant. The cut surfaces are fitted together and bound with raffia, scotch tape, or, preferably, with a self-sealing elastic bandage.

The leaf insert method of graft indexing, described by Bringhurst and Voth (1), has several advantages: no runners are required so that indexing can be done at any time of year; fewer donor plants are required for replicated indexing; and there is no danger of contaminating the donor plant with a latent virus from the indicator.



In leaf insert grafting, a leaf is detached from the donor plant. The side leaflets are removed and, if the leaf is large, most of the center leaflet cut off. The petiole is cut to wedge shape, half to three quarters of an inch long. On the indicator a center leaflet is removed from a young leaf and the petiole is split to correspond to the wedge on the donor leaf. The donor leaf is inserted snugly into this split and bound as for runner grafting. At least two leaflets should be inserted in each indicator. Survival of the inserted leaves for three weeks or more indicates successful graft union.

It should be emphasized that neither of these techniques, even where the union remains sound for many weeks, will assure transmission from an infected plant. Only repeated indexing will give any assurance that a given plant is virus-free.

Aphid transmission

The use of vectors in indexing is usually confined to those cases where the presence of a complex of viruses is suspected and it is desirable to separate the components.

Viruses that are aphid-transmissable may be separated from those that are not by the use of aphids. Furthermore, viruses that differ in their persistence may be separated from mixtures by varying the acquisition and transfer feeding periods, and by making serial transfers over a period of several days. Finally, certain aphid species may transmit one virus more efficiently than another and, by using selected vectors, individual viruses may be separated from a mixture.

The most important vectors of the strawberry viruses are members of the genus Pentatrichopus. Because of the difficulty of distinguishing between species of this genus, we have used clonal lines of aphids for most of our vector work in British Columbia.

A colony of virus-free aphids is easily established if two facts are born in mind: first that the young aphids do not acquire the virus directly from a viruliferous mother; and second that the nymphs do not start to feed for several hours after they are born. However, moving a newly born nymph without killing it is difficult. Our method of obtaining non-viruliferous colonies is as follows: an adult is transferred to a detached leaf and observed at intervals of one or two hours. As each nymph is born, the leaf piece on which it

rests is cut out and transferred to a fresh leaf from a virus-free plant. The nymphs move off the wilting leaf pieces before they start to feed and so will establish a clone that is certain to be virus-free.

Heat treatment

A number of strawberry viruses can be elminated by heat treatment. Mottle is the most heat-labile and is usually inactivated by growing infected plants at 100°F. for 2 weeks, a period which most varieties will withstand. Other viruses are eliminated only after such prolonged treatment that survival of the treated plant becomes uncertain.

Strawberry varieties differ in their ability to survive heat treatment, but in addition to inherent varietal differences, the condition of the plant, and the way it is handled during treatment, also affect survival. Plants should have well developed roots, even to the extent of being somewhat pot bound. Humidity should be low; 40 to 50% is adequate. Soil moisture should be kept to the minimum necessary to prevent wilting; overwatering, or allowing the pots to stand in water, will reduce survival. Evaporation from unprotected clay pots may reduce soil temperature as much as 10°F below the air temperature, and attempts to correct this by wrapping the pots in aluminum foil or plastic, or by using plastic pots, hasten plant mortality. According to Posnette and Cropley (22) removal of older leaves from the plants before treatment also shortens the period plants will survive.

It may be that plants will survive longer at a fluctuating temperature than at a constant temperature. The standard heat treatment is at a constant temperature of 100°F. We have had considerable success, however, using a heat chamber in which the daily temperature fluctuates from 95° to 115°F. Plants continue to grow actively in this chamber, and commonly survive for 3 or 4 months. One plant survived 8 months of treatment. We have succeeded in inactivating five viruses under these conditions. We have repeatedly eliminated mottle and latent-A viruses and, in a few instances, crinkle, witches' broom, and veinbanding.

The length of treatment required for virus elimination can be reduced by propagation of cuttings from treated plants. Posnette and Jha (24) describe propagation by slicing the crown of the treated plant into discs 0.5 or 1 cm. thick, and planting the discs in sand or peat. By this method they developed plants free of crinkle virus after only 2 or 3 weeks treatment whereas, in previous experiments, they had found a 50-day heat treatment necessary for the inactivation of this virus. Unfortunately, heat treatment decreased the proportion of cuttings that became established and, in one trial where 52 cuttings were made from plants treated for 2 weeks, only 5 survived.

We use another method of propagation. Small axillary buds are excised and rooted in sand under mist during, or very shortly after, heat treatment of the parent plants. By this method we have developed plants free of latent-A virus after 5 weeks treatment, whereas this virus is not eliminated from the parent plant by a 3-month heat treatment. Plants a year or more old are the most suitable for this type of treatment. On such plants, axillary buds frequently develop on the older part of the crown and can be removed during treatment as they reach the desired stage of develop-

ment. The smaller the bud excised, the more likely it is to be virus-free but buds weighing less than 20 mg. do not root readily. Furthermore, the development of at least a partially expanded leaf on the excised bud appears to be necessary for survival. The length of heat treatment has no adverse effect on rooting. We have established plants from buds excised after treatment periods of 4 months.

The apparent recovery of plants during heat treatment may be deceptive. Plants of the more heat-tolerant varieties, treated under optimum conditions, will continue to grow during treatment. On plants infected with viruses which cause symptoms, new leaves formed during treatment may be symptomless so that, with the death and removal of older leaves, the treated plants appear normal. After treatment, the re-appearance of symptoms may take much longer than expected on those plants in which the virus was almost, but not quite, inactivated. Plants developed from excised buds may remain symptomless even longer than the parent plants, and the smaller the excised bud, the longer the new plant will remain symptomless.

Posnette and Cropley (22) report that some plants infected with yellow edge virus remained symptomless for more than a year after treatment, and then symptoms reappeared.

We have found a similar, although less extreme, delay in the re-appearance of symptoms. In studying the elimination of latent-A virus by heat therapy, we used test plants infected with veinbanding and latent-A viruses, a combination which causes severe symptoms on commercial varieties, and from which elimination of latent-A is indicated by loss of symptoms from the treated plant. Some of the plants developed from excised buds appeared normal as long as 12 weeks before symptoms again appeared. All the plants that appeared normal at the end of that time, however, were still normal 3 years later, and it is assumed that latent-A was completely eliminated from these.

The reappearance of crinkle symptoms was similarly delayed on plants developed from excised buds. One plant, developed from an axillary bud, appeared normal for 4 months before crinkle symptoms reappeared, although the parent plant from which this bud was taken again showed symptoms 3 weeks after treatment.

With such experience it follows that, when latent viruses are involved, results should be interpreted with a great deal of caution. Indexing should be repeated over a long period of time before any treated plant is pronounced virus-free.

NOTE ON SOIL-BORNE VIRUSES INFECTING STRAWBERRY

A number of soil-borne viruses, whose principal hosts are other plants, have been shown to infect strawberry in Britain and Europe. With the exception of tobacco necrosis virus (6 and 11) there are no similar reports from North America, although the extent of survey has admittedly been limited.

Summarizing the situation in Britain, Lister (15) reports that all the isolates of soil-borne viruses infecting strawberry that were collected in England have proved to be strains of arabis mosaic virus, whereas almost

all collected in Scotland have been strains of tomato black ring and raspberry ringspot viruses.

The soil-borne viruses are not discussed in this review.

MOTTLE VIRUS, Thomas (30)

What is described here as strawberry mottle virus is evidently a group of viruses or virus strains which have similar vector characteristics and similar responses to heat therapy but which produce a very wide range of symptoms on Fragaria vesca.

SYNONYMS: Virus I (mild crinkle), Prentice and Harris (28)
Type I, Demaree and Marcus (3)
"Non-persistent component of yellows", Mellor and Fitzpatrick (18)

GEOGRAPHIC DISTRIBUTION: World wide.

SYMPTOMS: On commercial varieties there are no reliable symptoms but there is good evidence that some strains depress vigor.

On Fragaria vesca symptoms range from an almost undetectable mild mottling to severe stunting accompanied by various types of leaf distortions.

The first symptoms appear on the youngest leaf 10 to 20 days after inoculation. The petiole of this leaf is short, and one or two leaflets are reflexed and smaller than normal. These leaflets become mottled, crinkled, cupped, or otherwise distorted depending on the strain of the virus present. Subsequent leaves show the symptoms that are characteristic of that particular strain, although there may be minor fluctuations in their intensity depending on growing conditions and season of the year.

COMPLEXES WITH OTHER VIRUSES:

Mottle viruses combined with mild yellow edge virus produce xanthosis or yellows in susceptible commercial varieties and in F. vesca.

Mottle and latent-A viruses combined cause no diagnostic symptoms in commercial varieties, but vigor is depressed even further than by mottle alone.

Similarly, mottle and veinbanding combined cause no symptoms on the commercial varieties although again there is a greater depression of vigor than with either virus alone. In F. vesca, the mottle symptoms usually mask the veinbanding although the combined effect is greater than that of either virus alone.

TRANSMISSION is mainly by aphids of the genus Pentatrichopus: P. fragaefolii (Cock.), P. thomasi Ris Lambers, and P. thomasi ssp. jacobi Ris Lambers at the Pacific Coast; and P. minor (Forbes) in Eastern North America.

Aphids can acquire the virus from an infected plant within an hour but vector efficiency increases with longer feeding periods. The aphids usually lose their virus charge within 6 hours of leaving the source plant. There are differences in the efficiency of clonal lines of aphids, and the milder strains of the virus are sometimes difficult to transmit.

Other aphids that have been reported as vectors of mottle virus are: Acyrthosiphon malvae ssp. rogersii (Theob.), Amphorophora rubi (Kalt.), Aphis gossypii Glover, Macrosiphum pelargonii (Kalt.), Myzaphis rosarum (Walk.), Myzus ascalonicus Doncaster, M. ornatus Laing, M. porosus Sanderson, and Pentatrichopus tetrarhodus (Walk.).

HEAT INACTIVATION:

Mottle viruses can usually be eliminated by growing infected plants at 100°F for 10 to 14 days.

DETECTION AND IDENTIFICATION:

The East Malling strain of Fragaria vesca (E M C) is the most sensitive indicator for the mottle viruses. This strain is infected with strawberry latent-A virus which intensifies the symptoms of mottle.

Where the presence of other viruses is suspected, the plants should be heat-treated to eliminate the mottle viruses and allow the identification of any accompanying viruses.

REFERENCES: 3, 9, 16, 18, 19, 22, 23, 28.

ILLUSTRATIONS:

Fig. 1. Primary symptoms of mottle in East Malling F. vesca
Fig. 2. Chronic symptoms of mild mottle in East Malling F. vesca
Fig. 3. Chronic symptoms of moderately severe mottle in East Malling F. vesca
Fig. 4. Chronic symptoms of severe mottle in East Malling F. vesca (see also Figs. 5 and 6)

STRAWBERRY LATENT-A VIRUS, Frazier and Posnette (9)

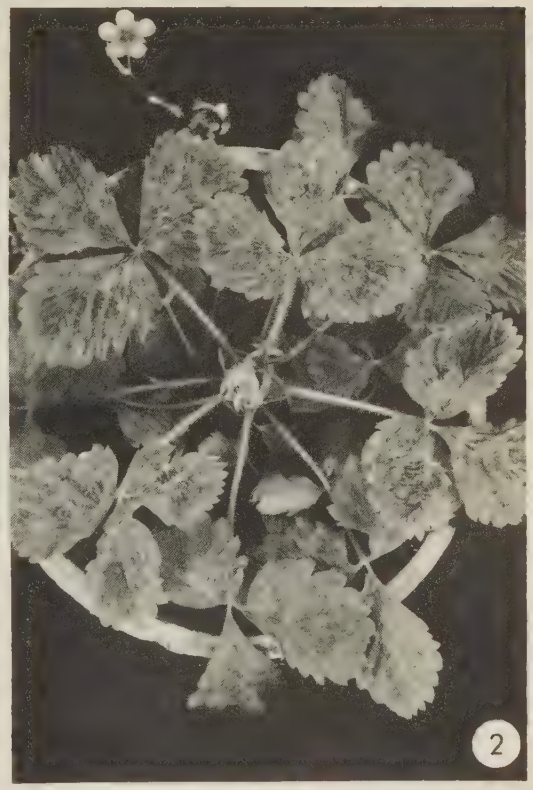
SYNONYMS: Strawberry latent virus, strain A, Frazier (4).

Note: According to Frazier and Posnette (9) this virus affords partial protection against the strawberry crinkle viruses and consequently can be considered as a strain of these. However, because of its importance, it is treated here as a separate virus.

GEOGRAPHIC DISTRIBUTION:

Little is known of the natural distribution of strawberry latent-A virus. It was originally found by Frazier in the strain of East Malling Fragaria vesca that Prentice and Harris had selected for its unusual sensitivity to the mottle viruses. Frazier also found a similar virus in a number of clones of F. californica growing in widely separated localities in the Coast Range mountains of California, and in a number of nursery colonies of apparently normal plants of the Marshall variety.


1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Wherever else it has been found, it is assumed to have been introduced into stock that has been indexed by runner grafting to East Malling F. vesca.

SYMPTOMS: Strawberry latent-A virus causes no symptoms, either on commercial varieties or on F. vesca. Its presence is detected by its influence on the symptoms of mottle or veinbanding viruses.

COMPLEXES WITH OTHER VIRUSES:

Latent-A and mottle viruses combined depress the vigor of commercial varieties but cause no tangible symptoms. In F. vesca, latent-A increases the severity of the symptoms of any particular mottle strain to those of a more severe strain.

Latent-A and veinbanding combined cause severe symptoms on commercial varieties and on F. vesca (see veinbanding virus).

TRANSMISSION is by grafting only; no vector has been found.

HEAT INACTIVATION:

Strawberry latent-A virus will survive in plants heat-treated for 3 months. It can be eliminated, however, by propagation of axillary buds excised from heat-treated plants. We have developed plants free of this virus from buds weighing up to 85 mg., excised after 5-weeks treatment, and from larger buds after longer treatment. Virus-free buds were excised up to 3 weeks after the end of the treatment.

DETECTION AND IDENTIFICATION:

Strawberry latent-A virus can be most easily detected by grafting to an indicator that is carrying veinbanding virus. The complex of the two viruses will produce the leaf distortion shown in Figs. 9 and 10.

REFERENCES: 4, 9, 16, 19.

ILLUSTRATIONS:

Fig. 5. Miller's latent-free F. vesca with mild mottle
Fig. 6. Miller's latent-free F. vesca with mild mottle and latent-A

(see also Figs. 8, 9 and 10)


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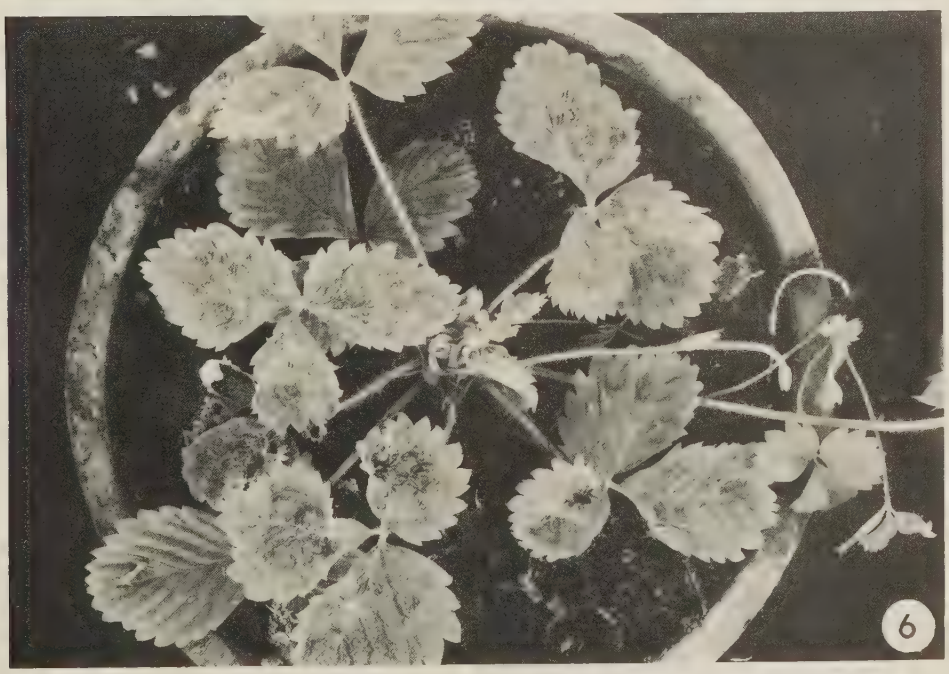
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VEINBANDING VIRUS, Frazier (5)

SYNONYMS: None, but the complex of a strain of veinbanding and strawberry latent-A virus in East Malling Fragaria vesca was described by Prentice as virus 5, or strawberry leaf curl virus (27).

GEOGRAPHIC DISTRIBUTION:

Occurs on both the Pacific and Atlantic Coasts of North America. It does not appear to occur naturally in Great Britain (Prentice's isolate was from a plant imported to England from the U.S.A.).

SYMPTOMS: On commercial varieties: There are no diagnostic symptoms although some strains may depress vigor slightly.

On Fragaria vesca: Symptoms appear 4 to 6 weeks after inoculation as discontinuous chlorotic streaks along the midribs and some secondary veins. Vigor is moderately reduced. The clarity of the veinbanding symptom fluctuates greatly and, particularly following transplanting or application of fertilizer, infected plants sometimes produce a series of symptomless leaves.

COMPLEXES WITH OTHER VIRUSES:

Veinbanding and latent-A viruses combined cause severe symptoms both on commercial varieties and on the indicators. On commercial varieties the leaves are twisted due to the shortening of portions of the veins. Chlorosis or purpling develops along the veins and dark purple lesions appear on the petioles. Vigor is much reduced. On F. vesca, the symptoms are similar but there is less purpling of the veins and more twisting of the leaves.

TRANSMISSION is by several aphid species representing a number of different genera. Vectors reported to date are: Amphorophora rubi (Kalt.), Aulacorthum solani (Kalt.), Aphis idaei van der Goot[4], Macrosiphum pelargonii (Kalt.), M. rosae (L.), Myzus ascalonicus Doncaster, M. ornatus Laing, M. persicae (Sulz.), Pentatrichopus fragaefolii (Cock.), P. tetrarhodus (Walk.), P. thomasi Ris Lambers, and P. thomasi spp. jacobi Ris Lambers.

Virus-vector relationships are similar to those of the mottle viruses. The aphids can acquire the virus from a source plant in 30 minutes but efficiency increases with longer feeds. Persistence in the vector is relatively short, being usually less than 6 hours. There are differences in the efficiency of clonal lines of aphids, and there is evidence that some aphid species will transmit some strains of veinbanding but not others (19).

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[4]The vector referred to by Mellor and Forbes (19) as Aphis rubifolii (Thomas) has since been identified as Aphis idaei van der Goot.

HEAT INACTIVATION:

Veinbanding is one of the more heat-stable viruses and its inactivation by heat has not previously been reported. We have found that it survived in plants treated for periods up to 8 months. We have one plant, however, from which veinbanding has been eliminated. This plant arose from an axillary bud after the parent plant was apparently killed by a 6-month heat treatment. Indexing this plant 18 months after the end of the treatment still failed to demonstrate the presence of the virus.

DETECTION AND IDENTIFICATION:

Since it is difficult to detect the veinbanding virus in the presence of strawberry mottle virus, plants to be indexed should first be heat-treated to remove the mottle viruses. The presence of veinbanding can then be demonstrated by transmission to indicators carrying latent-A virus, or to Alpine F. vesca.

REFERENCES: 5, 7, 9, 16, 19.

ILLUSTRATIONS:

Fig. 7. Veinbanding in Alpine F. vesca
Fig. 8. Veinbanding in Miller's latent-free F. vesca
Fig. 9. Veinbanding in East Malling F. vesca showing the downward curl and twisting of the leaflets characteristic of the veinbanding-latent A complex.
Fig. 10. Veinbanding-latent A complex in Marshall

MILD YELLOW EDGE VIRUS, Prentice (25)

SYNONYMS: Virus 2, Prentice (25)
"Persistent component of yellows" Mellor and Fitzpatrick (18)

GEOGRAPHIC DISTRIBUTION: Probably world wide.

SYMPTOMS: On commercial varieties: Because of the ubiquity of the strawberry mottle viruses, mild yellow edge seldom occurs alone under field conditions. It is virtually symptomless alone, causing little reduction in vigor and at most very slight chlorosis, mainly at the margins of the leaves.

On Fragaria vesca: Symptoms appear on plants of Frazier's U C 1 clone or on Alpine seedlings 4 to 6 weeks after inoculation, but may take much longer to appear on plants of the East Malling clone. Symptoms vary, depending on the strain of the indicator and on the strain of the virus. Plants of the East Malling clone are usually reduced in vigor with marginal chlorosis and slight cupping of the leaves. However, these symptoms are sometimes difficult to distinguish, even with healthy controls for comparison. Plants of Frazier's U C 1 clone, or Alpine seedlings, are better indicators for this virus. Symptoms on


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these indicators consist of premature reddening and yellowing of the older leaves, followed by scorching and their death.

COMPLEXES WITH OTHER VIRUSES:

Mild yellow edge and mottle viruses combined cause xanthosis or yellows in susceptible commercial varieties and in F. vesca. The economic importance of this disease in Europe and Western North America, and its lesser importance in Eastern North America are probably due to differences in the susceptibility of the different varieties grown in these areas rather than to the geographic distribution of the virus.

TRANSMISSION:

By aphids of the genus Pentatrichopus: P. fragaefolii, P. thomasi, and P. thomasi ssp. jacobi.

Aphids require acquisition and transfer feeding periods of 1 to 2 days each for transmission of this virus, and remain infective for 10 to 12 days after leaving the source plant.

HEAT INACTIVATION:

Mild yellow edge is not readily inactivated by heat therapy. Posnette and Cropley (22) report that plants were not cured by treatment at 37°C (98.5°F) for periods up to 26 days and although some plants remained symptomless for more than a year, they eventually relapsed. They did, however, obtain one runner plant, propagated immediately after treatment of the parent plant for 16 days, which was apparently virus-free two years later.

We have not eliminated this virus. Axillary buds, removed after treatment of the parent plant at 95-115°F for periods up to 3 months, were all infected.

DETECTION AND IDENTIFICATION:

Mild yellow edge can be separated from the xanthosis complex by allowing aphids to feed on source plants for several days, and then transferring them to a series of plants at daily intervals. The first plant or plants in the series will become infected with any non-persistent viruses (i.e. mottle and veinbanding) that may be present, whereas only mild yellow edge will be transmitted to plants further along the series.

The best diagnostic characters are probably the progressive reddening, yellowing, scorching, and dying of the older leaves on Frazier's U C 1 clone or on Alpine seedlings, and the ability to produce typical xanthosis when combined with mottle virus in a susceptible variety such as Marshall.

REFERENCES: 3, 9, 16, 18, 19, 22, 25.

ILLUSTRATIONS:

Fig. 11. Frazier's U C 1 F. vesca with mild yellow edge, showing the progressive scorching and dying of older leaves.

Fig. 12. Right: East Malling F. vesca with mild yellow edge
Left: East Malling F. vesca, control plant.

CRINKLE VIRUS Zeller and Vaughan (33)

SYNONYMS: Fragaria virus 2, Zeller and Vaughan
Virus 3, Prentice (26)
Strawberry virus 4, J. Johnson
Marmor fragariae, Holmes.

GEOGRAPHIC DISTRIBUTION: Pacific coast of North America, and Great Britain.

SYMPTOMS: On commercial varieties: The more severe strains of crinkle virus seriously reduce the vigor of plants of susceptible varieties such as Marshall. Yellow spots appear on the leaves, varying in size from pinpoints to large chlorotic areas which cause leaf distortion. Leaflets are unequal in size and there is marked marginal chlorosis of the distorted leaves. Very mild strains of the virus may cause only small chlorotic sectors and, occasionally, unequal size of leaflets. On varieties less susceptible than Marshall, symptoms may be so mild that indexing is required for diagnosis.

On Fragaria vesca: On plants of Frazier's U C 1 clone or on Alpine seedlings, symptoms appear about 4 weeks after graft inoculation. On plants of the East Malling clone, symptoms are much slower to develop,


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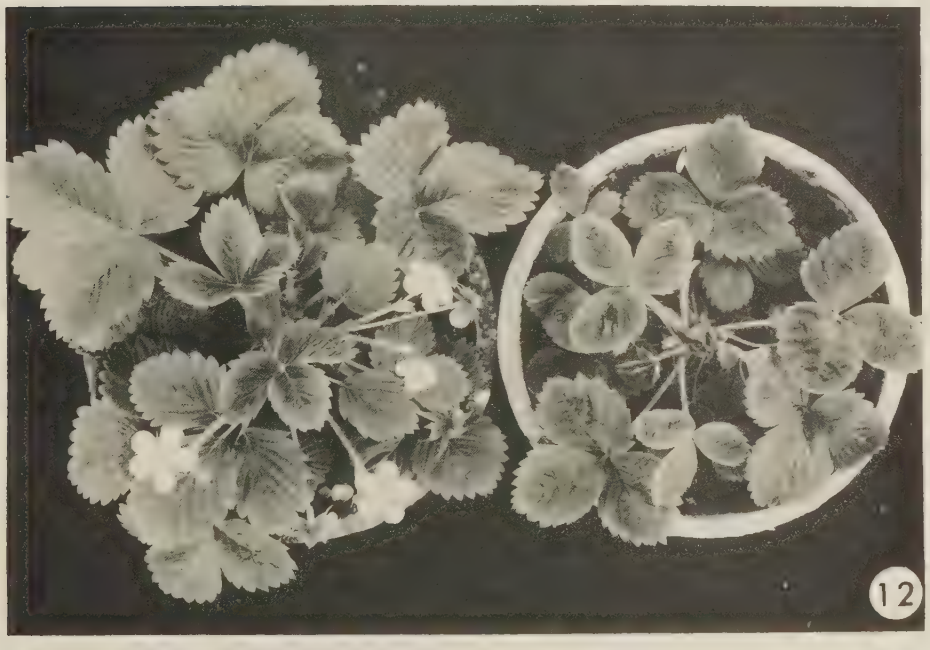
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presumably because the latent-A virus affords partial protection against infection by the crinkle virus. Symptoms are similar on the 3 indicators. Pinpoint chlorotic spots appear which may, in severe cases, cause leaf distortion. Lesions frequently appear on petioles and stolons, sometimes causing the angular bending shown in Fig. 15. Very mild strains may cause only petiole lesions and, occasionally, the backward bending of a center leaflet.

COMPLEXES WITH OTHER VIRUSES:

Crinkle and mottle viruses together seriously reduce the vigor of plants of the Marshall variety; crinkle, mottle, and mild yellow edge together cause very severe degeneration; and if latent-A is also present the degeneration is even more extreme. These latter complexes will even reduce the vigor and fruitfulness of varieties such as Northwest, which are highly tolerant of the mottle-mild yellow edge complex, and will cause speckling and mild chlorosis.

TRANSMISSION:

The virus-vector relationships of the crinkle virus and its strain are not clearly understood. Vaughan (31), and Prentice (26) report transmission of crinkle by the strawberry aphid without any particular difficulty. More recent attempts by Frazier and Posnette in England (9), and Mellor and Forbes in British Columbia (19), have met with indifferent success or downright failure. Naturally infective aphids, taken from diseased plants in the field, transmitted the virus (9), but no transmissions were obtained by aphids reared in the laboratory and used in controlled experiments.

The most that can be said at the present time is that the strawberry aphids, Pentatrichopus fragaefolii certainly, and probably P. thomasi also, are the principal vectors. According to Prentice and Woollcombe (29) the aphids can acquire the virus within 24 hours, but an incubation period of 12 to 16 days is required before they can transmit. The insects retain their ability to transmit for several days.

HEAT INACTIVATION:

Posnette and Cropley (22) report elimination of crinkle virus by treatments at 37°C (98.5°F) for 50 days. They found some strains easier to inactivate than others. Posnette and Jha (24) developed crinkle-free plants from stem cuttings taken after 2 or 3 weeks heat treatment of the parent plant. We have developed a crinkle-free plant from an axillary bud excised from an infected plant after 8-weeks heat treatment. The parent plant, however, remained infected.

DETECTION AND IDENTIFICATION:

On commercial varieties crinkle symptoms may be confused with other disorders. For example, the complex of veinbanding and latent-A viruses, or infestations of the shallot aphid (Myzus ascalonicus) or of two-spotted mite, produce symptoms that may be confused with crinkle. However, if the symptoms are caused by the veinbanding-latent-A complex, and no

crinkle is involved, grafting to F. vesca will give the distinctive symptoms of this complex (see veinbanding virus). If the trouble is from the shallot aphid, close examination early in the season will reveal the aphids, and the plants will recover completely as new leaves are formed during the summer. If symptoms are due to mite infestation, closer examination will reveal the mites, and leaves formed after elimination of the mites will be normal.

On F. vesca the leaf symptoms of crinkle virus are nearly indistinguishable from those caused by some strains of mottle virus on East Malling F. vesca. This was the source of much of the early confusion between so-called "mild crinkle" (i.e. mottle) and bona fide crinkle. Mottle, however, does not cause the petiole and stolon lesions.

REFERENCES: 9, 19, 22, 24, 26, 29, 31, 33.

ILLUSTRATIONS:

Fig. 13. Marshall with severe crinkle.
Fig. 14. Frazier's U C 1 with crinkle, showing leaf symptoms and petiole lesions.
Fig. 15. Frazier's U C 1 with crinkle, showing lesions which cause sharp bending of the stolon.

LATENT-C VIRUS, McGrew (16)

SYNONYMS: The complex of latent-C and latent-A viruses in East Malling Fragaria vesca was described by Demaree and Marcus (3) as "type 2 symptoms".

GEOGRAPHIC DISTRIBUTION: Eastern North America.

SYMPTOMS: On commercial varieties latent-C virus causes no symptoms, but it decreases vigor of at least some varieties.

On Fragaria vesca of the East Malling clone, Fulton's latent-free clone, and on some seedlings of the East Malling clone, latent-C causes a shock symptom consisting of severe epinasty of the young leaves. This is followed by a proliferation of crowns with very small leaves. On Alpine F. vesca, Frazier's U C 1 clone, Miller's latent-free clone, and on some seedlings of the East Malling clone, latent-C causes no symptoms, but it decreases vigor.

TRANSMISSION is by graft only; no vector has been reported.

HEAT INACTIVATION:

All attempts to free plants of this virus by heat therapy have so far failed.

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DETECTION AND IDENTIFICATION:

After mottle has been eliminated by heat therapy, latent-C can be detected by graft-inoculation of East Malling F. vesca. On this indicator latent-C symptoms could only be confused with those of a complex of witches' broom and veinbanding. However the witches' broom virus, with or without veinbanding, causes crown proliferation on commercial varieties and on all the common indicator species, while latent-C does not cause crown proliferation on commercial varieties, on Alpine, on Miller's latent-free clone, or on Frazier's U C 1.

REFERENCES: 3, 16, 17.

ILLUSTRATIONS:

Fig. 16. Primary symptoms of latent-C in East Malling F. vesca.
Fig. 17. Chronic symptoms of latent-C in East Malling F. vesca.

WITCHES' BROOM VIRUS, Zeller (32)

SYNONYMS: Strawberry virus 2, J. Johnson
Fragaria virus 3, Zeller
Blastogenus fragariae, McKinney
Nanus fragaria, Holmes

GEOGRAPHIC DISTRIBUTION: North America

SYMPTOMS: On commercial varieties: Plants are dwarfed, "bushy" in appearance, with multibranched crowns, and erect, spindly petioles supporting small leaves.

On Fragaria vesca symptoms are similar to those on the commercial varieties.

TRANSMISSION:

Zeller (32) reported transmission by the strawberry aphid, Pentatrichopus sp. Mellor and Forbes (19) have been unable to obtain transmission with either P. fragaefolii or P. thomasi.

HEAT INACTIVATION:

We have eliminated this virus from a few plants of the British Sovereign variety by growing them at 95-115° F for 8 or 10 weeks.

DETECTION AND IDENTIFICATION:

On commercial varieties the field symptoms are distinctive and there is usually no difficulty in identifying the disease.

On F. vesca the symptoms are similar to chronic symptoms of latent-C virus. However, latent-C does not produce witches' broom in commercial varieties. Since, in experiments with Premier known to be carrying latent-C, there was no protection against witches' broom, it must be assumed that the two viruses are not related.


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REFERENCES: 19, 20, 32.

ILLUSTRATIONS:

Fig. 18. Witches' broom in British Sovereign
Fig. 19. Witches' broom in East Malling F. vesca

ASTER YELLOWS VIRUS, Kunkel

SYNONYMS: Western aster yellows virus
Callistephus virus 1A Smith
Chlorogenus callistephi var. californicus, Holmes.

GEOGRAPHIC DISTRIBUTION: California, Arkansas.

SYMPTOMS: Phyllody of the flowers of infected strawberry plants is similar to the symptom typical of this virus on other plants. Plants eventually die.

TRANSMISSION is by leafhoppers (Macrosteles fascifrons, Colladonus geminatus, and C. montanus) but with considerable difficulty.

HEAT INACTIVATION: No information.

DETECTION AND IDENTIFICATION: See symptoms.

REFERENCES: 9, 10, 12.

GREEN PETAL VIRUS, Posnette (21)

SYNONYMS: None. Frazier and Posnette (8) state that variations in symptoms on strawberry plants suggest that two diseases may be grouped under the name "green petal." They distinguish these as (a) green petal caused by the virus inducing phyllody in clover, and (b) bronze leaf wilt caused by the clover witches' broom virus. They also suggest that green petal virus may be related to aster yellows virus.

GEOGRAPHIC DISTRIBUTION: England, Eastern Canada.

SYMPTOMS: The flower symptoms are the most characteristic of this disease, and these may appear while the foliage still remains normal. Sepals are enlarged; petals dwarfed and pale green. Some flowers are sterile; others form a small, hard, green receptacle which fails to ripen and from which the achenes stand out, appearing unusually large. Dried inflorescences are a useful symptom in field diagnosis.

Leaves formed after infection are dwarfed, slightly cupped, with main veins and margins yellow. The old leaves first turn dull yellowish or olive green and then bright red in August and September. The whole plant may collapse and die in mid-summer or there may be temporary recovery in

September with the formation of secondary crowns with minute leaves. The stolons are short and thickened, and the young runner plants extremely dwarfed.

TRANSMISSION is by graft; not by the strawberry aphid. The virus has been transmitted from strawberry to clover by leafhoppers.

HEAT INACTIVATION: No information.

DETECTION AND IDENTIFICATION: See symptoms.

REFERENCES: 2, 8, 9, 13, 21.

ILLUSTRATIONS:

Fig. 20. Flower symptoms of green petal virus in contrast to normal flowers, on field plants of the variety Senator Dunlap.
Fig. 21. Flower symptoms of green petal virus.
Fig. 22. Fruit symptoms of green petal virus.


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CHARACTERISTICS OF STRAWBERRY VIRUSES

Virus	Symptoms					Vectors	Inactivation by heat
	Commercial varieties	F. vesca clones					
		EMC	Alpine	UC 1	EMK		
Mottle	-	S	S	S	S	aphids NP	A
Latent-A	-	-	-	-	-	unknown	B
Veinbanding	-	S	S	S	S	aphids NP	C
Mild yellow edge	-	S	S	S	S	aphids P	C
Crinkle	S	S	S	S		aphids P	B
Latent-C	-	S	-	-	S	unknown	D
Witches' broom	S	S	S	S	S	aphids ?	B
Aster yellows	S					leafhoppers	
Green petal	S					leafhoppers	

Key to Symbols

Symptoms: S indicates diagnostic symptoms; - indicates no diagnostic symptoms

Vectors: NP non-persistent in vector; P persistent in vector

Inactivation by heat: A readily inactivated.
B inactivated only by prolonged heat treatment, or by taking cuttings from heat treated plants.
C inactivation in a single instance.
D has not yet been inactivated.

Summary

The detection and identification of latent viruses in commercial strawberry plants usually requires three steps: preliminary indexing to determine whether the mottle viruses are present, heat treatment to eliminate mottle, and re-indexing the heat-treated plants to detect any heat-stable viruses. For the re-indexing, three indicators are desirable: East Malling F. vesca to show veinbanding or latent-C (or mottle), U C 1 or Alpine to show mild yellow edge, and one of the latent-free F. vesca clones infected with veinbanding, to show latent-A. When the results of indexing are negative the tests should be repeated several times before any plant is pronounced virus-free.

Acknowledgements

Most of the information given in this review is either from published data or from our own experience. Much of the information on crinkle virus, however, was supplied by Dr. C.D. Schwartze of the Western Washington Experiment Station, Puyallup, Washington, and the illustrations of the symptoms of green petal virus were provided by Mr. C.O. Gourley (Fig. 20), of the Canada Agriculture Research Station at Kentville, Nova Scotia, and Dr. R.O. Lachance (Figs. 21 and 22) of the Canada Agriculture Research Laboratory at Ste. Anne de la Pocatiere, Quebec.

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A REVIEW OF THE TAXONOMY AND NOMENCLATURE OF SOME LOW-TEMPERATURE FORAGE PATHOGENS[1/]

W.C. McDonald[2/]

At the 1960 meeting of the Diseases of Forage Crops Sub-Committee of the Associate Committee on Plant Diseases it was suggested that a study be made of the taxonomy of low-temperature fungi attacking forage crops, with particular reference to the genera Typhula and Sclerotinia. This suggestion was forwarded by Dr. J.B. Lebeau, C.D.A. Research Station, Lethbridge, on behalf of a group of plant pathologists attending the 9th International Botanical Congress, Montreal, who were disturbed over the confusion created in the literature by the use of several specific epithets for fungi which were probably identical morphologically. A project on this topic was assigned to the Winnipeg laboratory for study. This report consists of a critical review of the literature which was made as the first step in the investigation.

SCLEROTINIA

Sclerotinia borealis Bub. & Vleug., in Vleugel, Svensk Bot. Tidskr. 11: 308. 1917.
Synonym - Sclerotinia graminearum Elen. ex Solkina, Pl. Prot., Leningr. 18: 100-108. 1939. (R.A.M. 18: 582).

The main point of confusion in naming low-temperature pathogens in this genus is the use of the name S. graminearum in Russia (26) and Japan (23) and S. borealis in the rest of the world (6, 11, 13, 17, 20) for what most people believe to be the same fungus. Jamalainen (11) reviewed the literature concerning the taxonomy and suggests the probable synonymy of the two species. Solkina in 1939 (19) compared specimens of S. graminearum with related species and, although she retained the name S. graminearum for her Russian isolates, did indicate the similarity between them and S. borealis. The description given by Solkina for S. graminearum agrees sufficiently well with that given by Groves and Bowerman (7) for S. borealis that they can be considered to be the same species. Recently Sprague et al (20) compared specimens of S. borealis collected in the U.S.A. with the description of S. graminearum and arrived at the same conclusion. As the name S. borealis has priority over S. graminearum it should be adopted as the correct name for the fungus.

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TYPHULA

Typhula incarnata Lasch ex Fr., Epicr., 585, 1838.
Synonyms: T. itoana Imai, Trans. Sapporo Nat. Hist. Soc. 11:39-44. 1929.
T. graminum auctt. non Karst. Volk, Zeitschr. f. Pfl. Krankh. 47: 339. 1937.

T. incarnata, according to Corner (2), has priority over T. itoana which is commonly used (1, 4, 12, 17, 18). In Japan and the U.S.A. the binomial T. incarnata has been accepted (20, 24). Isolates of this species have been misidentified as T. graminum Karst. (25) but Imai (10) showed that the two species were different. There seems to be no reason why T. incarnata cannot be accepted as the correct name of the species.

Typhula graminum Karst., Finl. Nat. o. Folk 37: 183. 1882.

The prevalence of this species on grasses is difficult to determine because of the confusion in the literature between it and T. incarnata. Reports of its occurrence without a description of the fungus leaves doubt as to the species actually encountered. Corner (2) believes that T. graminum is probably not uncommon but that it is very small and inconspicuous.

Typhula ishikariensis Imai, Trans. Sapporo Nat. Hist. Soc. 11:74. 1930.
Synonyms: T. idahoensis Remsberg, Mycologia 32: 89. 1940.
T. borealis Ekstrand, Medd. Växtskyddsanst., Stockh. 67. 125 pp. 1955.
T. hyperborea Ekstrand, Medd. Växtskyddsanst., Stockh. 67. 125 pp. 1955.

Imai described T. ishikariensis in Japan from specimens collected during October and November on rotting stalks or petioles of Trifolium pratense L. and rotting leaves and culms of wheat and grasses (9). It is not as widely distributed in Japan as T. incarnata (21) and was ignored until Tomiyama's work (21, 22, 23, 24) on the comparative pathogenicity of the two species. Neither Remsberg nor Corner mention this species in their monographs nor does Ekstrand refer to it in his papers. Remsberg described T. idahoensis in the U.S.A. in 1940 (15) three years after Ekstrand (3) in Sweden differentiated a species, T. borealis, from T. incarnata but did not publish a description of the fruit bodies of his fungus. Ekstrand has maintained that his isolates do not fit Remsberg's description of T. idahoensis and in 1955 (5) divided his original species into two species. T. borealis and T. hyperborea, on the basis of the length-width ratio of their basidiospores; they are indistinguishable otherwise. Some doubts as to the realiability of such a diagnostic character is suggested by Ekstrand's statement (5) that: "The length of the spores is very variable in different fruit bodies and in different collections. The relation between the length and width of the spores is very variable too, due to the variation in the length which is very great even in the spores of fruit bodies from the same collection". T. borealis has been identified by Ekstrand (6) on material collected in Sweden, Finland Canada, and Norway. Jamalainen (12) has since acknowledged that isolates

of Typhula occurring in Finland agree with the description of T. idahoensis and should be referred to that species. Corner (2) and Remsberg (16) consider that T. borealis is synonymous with T. idahoensis.

The description given by Imai (9) for T. ishikariensis agrees fairly well with those given by Remsberg (15) for T. idahoensis and Ekstrand (5) for T. borealis, considering the variability of the fungus. A comparison of the taxonomic features taken from descriptions of (A), T. idahoensis; (B), T. borealis; and (C), T. ishikariensis are tabulated below:

Sclerotia -(A) globose, flattened; chestnut-brown to blackish; 0.5-0.9 x 1-2 mm.
(B) rounded to subglobose; brown to black; up to 1.5 mm diam.
(C) globose, ellipsoid, often compressed; dark-brown to black; 0.5-1 mm.

Clavula -(A) cylindrical, elongate, fusiform; vinaceous-brown to leathery-brown; 4-7 mm long, 0.5-1.5 mm thick.
(B) clavate or cylindrical; white to brown-velvet; stipe and clavula variable in length -together up to 30 mm.
(C) cylindrical, oblong-clavate, fusiform; white or whitish becoming light yellowish-brown when dried; 2-5 mm long, 0.5-1.0 mm thick.

Stipe -(A) distinct; bistre, umber, or dark-brown; 2.5 x 0.1-0.5 mm.
(B) distinct; lighter or darker grey-brown, darker towards sclerotium.
(C) distinctly marked off from clavula; brownish; 3-10 mm.

Basidia -(A) elongate, thicker at apex; 5.8-7.8 μ thick; 4-6-8 spored.
(B) 4-spored
(C) cylindrical, clavate; 5.5 μ thick; 4-spored

Spores -(A) ovate-ellipsoid, apiculate; 8-14 x 3.8-7.8 μ, aver. 10.5 x 4.5 μ.
(B) ovate-oblong to ovate-subcylindric; 5.5-13.25 x 2.0-4.5 μ, aver. 8.9 x 3.2 μ. (T. hyperborea - 5.5-11 x 2.75-5.75 μ. aver. 8.4 x 4.3 μ)
(C) ellipsoid or oblong; 8-10 x 4 μ.

Hosts -(A) wheat, grasses, clover?(14), not pathogenic to alfalfa (1).
(B) cereals, grasses, clover, alfalfa, rape, beets and many other kinds of plants from different families.
(C) cereals, clover, rape (24).

It is unfortunate that the structure of the sclerotia of T. ishikariensis and T. borealis has not been described because both Corner (2) and Remsberg (15) believe it to be of primary diagnostic importance in the taxonomy of the genus.

The descriptions of the four fungi (T. hyperborea is identical with T. borealis except for spore size) appear to be sufficiently similar to justify the belief that they are co-specific. As the name T. ishikariensis has priority the other names must be placed in synonomy.

Conclusion

The work involved in collected a sufficiently large number of specimens of Sclerotinia spp. and Typhula spp. from widely separated geographic areas for comparative study does not seem warranted on the basis of what is already known about them. However, some doubts may still exist as to the correctness of placing certain names in synonymy with T. ishikariensis. These will not be resolved until Japanese, American, and Swedish specimens are compared culturally, morphologically, and pathogenically by one investigator.

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VARIATION IN ISOLATES OF DIDYMELLA APPLANATA[1]

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Introduction

During the years from 1949 to 1957, considerable variation was observed in the susceptibility of commercial raspberry varieties to spur blight, caused by Didymella applanata (Niessl) Sacc. Most of the varieties grown in Ontario and Quebec exhibited moderate to severe symptoms in years when there were prolonged humid conditions during May and early June. Isolations made from spur blight lesions on these varieties yielded a single cultural type of the organism which remained quite stable in culture. During this time, the variety Newburgh was grown beside the severely infected varieties without becoming infected. However, in 1958, Newburgh, growing beside the susceptible Rideau variety, became severely infected and developed typical spur blight lesions. This observation led to work on determining variation in pathogenicity.

Cultural differences among 3 isolates

D. applanata was isolated from commercial and wild raspberries growing in Ontario and Quebec. Over 50 isolations from 22 localities yielded 3 fairly distinct cultural types. Differences in colony type and in microscopic characters were observed in culture. An isolate from wild raspberries, referred to as isolate #1 in this paper, produced a spreading depressed type of growth with abundant pycnidia produced in clumps (Fig. 1A). The medium (P.D.A.) became slightly discolored in 10 days. This isolate was fast-growing and covered a petri plate in 28 days.

Isolate #2, from the commercial varieties, Trent, Rideau, Madawaska, Muskoka, Viking, September, Tweed, Latham, Carnival, Gatineau, and Ottawa yielded colonies that were more limited in lateral spread, produced more abundant aerial mycelium, and fewer pycnidia. This isolate also discolored the medium (Fig. 1B).

Isolate #3, from the variety Newburgh, produced a colony quite different from the other two. Very little aerial mycelium was produced. Pycnidia were produced in great quantity giving the colony a very dark appearance. The medium remained uncolored (Fig. 1C).

Isolates #1 and #2 produced pycnidia in 10 days. These were similar in color and wall consistency, being light-brown at first, and becoming black after 25 days. The walls were quite soft and did not become brittle until the cultures were 4 weeks old. Pycnidia produced by isolate #2 were considerably larger and more globose than those formed by isolate #1 (Table 1). Conidia produced by the 2 isolates fell in approximately the same size range, but the average size of those of isolate #2 was slightly larger.

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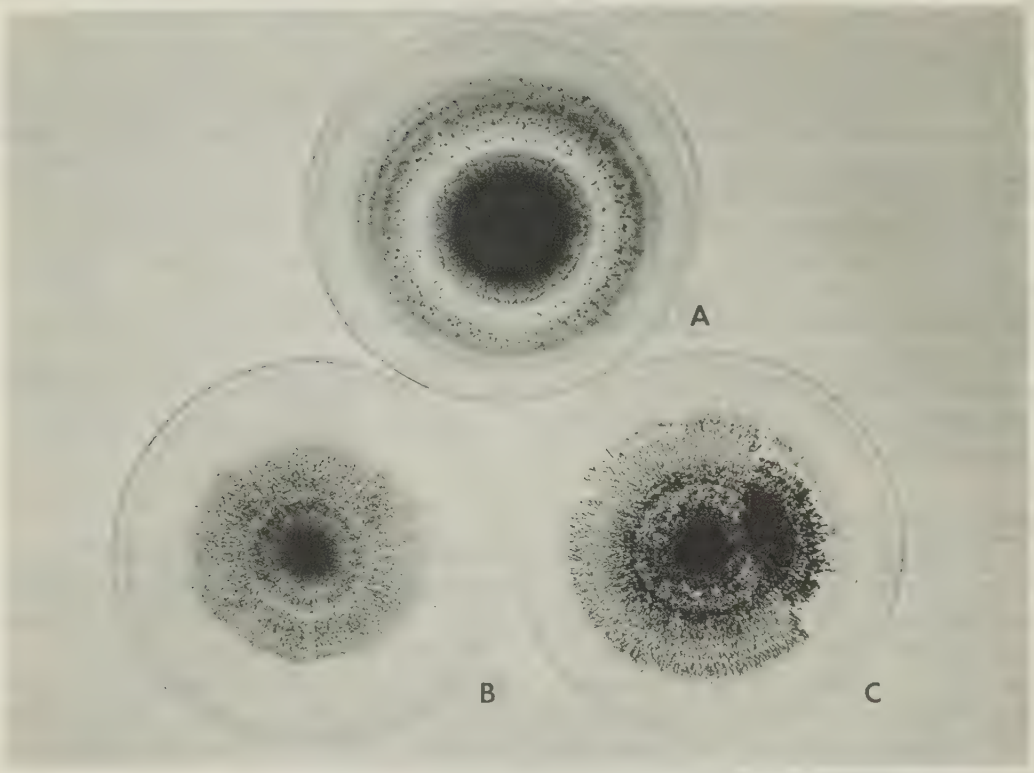

Fig. 1. A–Isolate #1 B–Isolate #2 C–Isolate #3

Isolate #3 produced large numbers of pycnidia after 5 days. These were much more numerous than in cultures of the other isolates, and were considerably smaller (Table 1). They were dark in color and brittle in wall texture at this time. The conidia were considerably larger than those of the other 2 isolates and were exuded more freely onto the surface of the medium. The relative differences in measurements of pycnidia and conidia were also evident on the hosts (Table 2).

Table 1. Size and number of pycnidia and size of conidia produced on P.D.A. 3 isolates of D. applanata (28-day old cultures)

Isolate	Number of pycnidia per sq. cm.	Size of pycnidia (μ) range	Size of pycnidia (μ) average	Size of conidia (μ) range	Size of conidia (μ) average
#1	120	85-190x135-255	135x190	2.7-3.4x6.0-6.7	2.8x6.4
#2	40	205-340x235-340	225x250	2.7-4.0x4.7-6.7	3.3x6.6
#3	1400	75-185x 85-210	105x135	2.0-4.0x6.0-13.4	3.7x8.1

Table 2. Size of pycnidia and conidia produced on raspberry canes after inoculation with 3 isolates of D. applanata.

Isolate	Size of pycnidia (u) range	Size of pycnidia (u) average	Size of conidia (u) range	Size of conidia (u) average
#1	110-225x160-275	160x210	2.7-3.8x6.0-7.2	3.1x6.7
#2	205-380x240-370	240x260	2.8-4.0x5.2-7.4	3.4x6.8
#3	75-195x105-250	115x150	2.6-4.5x6.5-14.0	3.7x8.4

Differences in life cycle

Several canes of the varieties Trent, Madawaska, Rideau and Newburgh, growing in pots, were inoculated with a conidial suspension of D. applanata. Isolates #1 and #2 produced severe lesions on the canes of Trent, Madawaska, and Rideau. Isolate #3 produced a few lesions on all varieties. The plants were placed outdoors over winter and examined in the spring. The isolates were recovered from their respective hosts. Perithecia were found on the canes inoculated with isolates #1 and #2. Only pycnidia were produced on the canes inoculated with isolate #3. It is probable that this isolate reproduces in nature by conidia only and does not employ the perfect stage in its life cycle. Perithecia were found in large quantities in the field on all varieties except Newburgh. Cultures from these were always of the type of isolate #2.

Pathogenicity

All 3 isolates were capable of causing infection on raspberry canes. Canes of the varieties Newburgh, Carnival, Madawaska, Trent, and Latham were infected by the 3 isolates when the bark was damaged and a conidial suspension introduced by means of a hypodermic needle. When the conidia were applied to uninjured canes and leaves with an atomizer, the results shown in Table 3 were observed. Isolate #3 caused severe leaf lesions and defoliation on the 5 varieties, whereas very few leaf lesions were produced after inoculation with isolates #1 and #2.

Table 3. Results of inoculating 5 raspberry varieties with conidial suspensions of 3 isolates of D. applanata.

Inoculum used	Newburgh	Carnival	Madawaska	Trent	Latham
Isolate #1	0	S	S	S	S
Isolate #2	0	M	S	S	S
Isolate #3	S	S	S	S	S

0 = no infection M = moderate infection S = severe infection

From Table 3, it can be concluded that isolates #1 and #2 were different from isolate #3 in ability to infect Newburgh. This difference in pathogenicity leads one to belive that isolate #3 constitutes a different race or possibly a new form since there are differences both in pathogenicity and morphology.

Discussion

Races of the fungus, D. applanata must be considered in breeding for spur blight resistance in raspberries. Since isolations made from wild raspberries in several localities in Ontario and Quebec yielded only one biotype, it is quite likely that, among the wild plants in this area, only one race occurs. Since no differences in pathogenicity were observed between this isolate and the common isolate from commercial varieties, isolates #1 and #2 described above must be considered to represent a single race. Newburgh, being resistant to this race, probably contains one or more genes for resistance to it, but not to the race described in this paper as isolate #3. It would seem, then, that additional races of the fungus may be found among isolates of D. applanata from other parts of the world where numerous varieties of red raspberry are grown. Work is being continued on this phase of the problem.

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CURLY-TOP, A VIRUS DISEASE OF FLORISTS' GERANIUM IN ONTARIO[1]

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Abstract

Evidence is presented to prove that the pronounced yellow veinbanding and curling, observed on the foliage of Pelargonium hortorum var. "George Tassel" at Dunnville, Ontario, is of virus origin.

Introduction

In December, 1958, peculiar symptoms suggestive of virus infection were observed in two three-year old stock plants of Pelargonium hortorum Bailey var. "George Tassel" in a greenhouse at Dunnville, Ontario. The syndrome in this variety was unlike that of any of the virus diseases of geranium hitherto reported in Canada. This report describes the disease and presents some experimental evidence concerning its nature.

Symptoms

Affected plants of the variety George Tassel develop a pronounced yellowing of the entire network of veins of many of the leaves under conditions of low temperature and low light. Some of the foliage on three-year-old plants appears mildly chlorotic. The younger, vein-cleared leaves are cupped and frequently curled downward (Fig. 1). On the older, woody parts of the plant, extremely small, curled, vein-cleared leaves develop from many of the nodes. By March, 1959, both of the affected plants and their progeny lost the marked vein-clearing. Though this feature of the disease was still faintly discernible at that time, most new growth showed only moderate chlorosis with no cupping or curling. Conspicuous symptoms reappeared during January, 1960, under greenhouse conditions.

Transmission

Scions from healthy seedlings of Pelargonium zonale Ait. were top grafted to affected George Tassel plants to determine whether the symptom complex was graft-transmissible.

Eighteen such graft combinations were made during the first week of December, 1959. The grafted plants were kept under constant surveillance for four months. Three scions showed mild but distinct veinal chlorosis but no curling of the leaves in 21 days. Two weeks later, an additional seven scions exhibited these mild symptoms. Not until two months after grafting was pro-

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Fig. 1. Symptoms of curly-top on the geranium variety George Tassel.

nounced yellow vein-clearing observed. These more severe symptoms appeared on only three leaves of a single scion. Definite cupping of the foliage was found, however, on three previously healthy scions. As summer approached, the symptoms became less and less apparent. They recurred in January, 1960, and again in January, 1961, on a few plants maintained in the greenhouse.

During January, 1960, three symptomless, presumably healthy, George Tassel scions were top-grafted to affected plants. Marked vein-banding and some downward cupping was noted on one of the scions in January 1961. No definite symptoms were apparent before this time.

In the first year following grafting, none of the scions exhibited the severe curly top symptom that seems to be associated with the advanced stage of infection in older plants. More pronounced symptoms that approached those found on the naturally affected George Tassel appeared, however, in the latter part of the second year.

Mechanical inoculations by conventional methods, using affected leaves of George Tassel as inoculum, failed to infect Pelargonium zonale Ait., Nicotiana glutinosa L., N. tabacum L., N. rustica L., Datura stramonium L., Chenopodium amaranticolor Coste and Reign., Cucumis sativa L., and Gomphrena globosa L.

Neither a bacterial nor a fungous pathogen was isolated from stem tissue of geraniums affected by curly-top.

Discussion and Conclusions

The experimental evidence indicates that the disease syndrome observed in plants of the variety George Tassel is of virus origin. The results do not exclude the possibility that curly-top in Ontario is a composite disease caused by multiple virus infection. However, the affected plants, as originally observed, have never shown the characteristic symptoms of leaf curl (2) and mosaic (4, 5), the only virus diseases of geranium reported in Canada. Nevertheless, the viruses responsible for these diseases might be masked in George Tassel under the environmental conditions in our greenhouses. Moreover, other latent, undetected viruses might be present and could account in part for the symptoms noted. Since the common geranium viruses are not usually mechanically transmissible and infect few plant species, the separation of component viruses in any complex in this host is prevented.

The identity of curly-top and its relationship to other virus diseases described elsewhere in geranium has of necessity been based on symptoms alone. In some characteristics, the Ontario disease resembles and could be similar to the curly-top of geranium that occurs in California. Severin and Freitag (7) described this disease in 1934 and showed that it was caused by Beta virus 1, which is transmitted by the beet leafhopper, Circulifer tenellus (Baker) (=Euttetix tenellus (Baker) of Severin and Freitag). However, no protuberances such as they described were observed on the veins of the undersides of the leaves of affected George Tassel. McWhorter (6) described a geranium disease that he called leaf cupping. There appeared to be some question in his mind as to whether curly-top and leaf cupping should be considered the same, though he felt that Severin and Freitag had clearly showed that the curly-top virus caused a leaf cupping disease of Pelargonium. Moreover, he believed that they confused other troubles with accepted curly-top symptoms. The protuberances described by them have been shown to be the direct result of insect feeding, not a virus. This fact might account for my failure to find this symptom on my infected plants. Insects have never been observed on the naturally infected plants, nor have they been seen on the graft-inoculated scions.

Kivilaan and Scheffer (3) also described a leaf cupping disease of geranium that they attributed to the curly-top virus. Their illustration showing leaf cupping in a seedling plant grafted with a scion from a symptomless plant does not resemble the Ontario disease as observed in naturally infected George Tassel, graft-inoculated, presumably healthy, George Tassel, or grafted seedlings of P. zonale.

Inasmuch as the symptoms of the Ontario disease do not agree in all respects with those reported for curly-top in California and leaf-cupping in Michigan, it seems best at present not to identify the Ontario disease with either of the two until their relationships have been further investigated.

Should this disease in fact be caused by Beta virus 1, concern about its natural spread in Ontario can be dismissed because the only insect vector of Beta virus 1, the beet leafhopper, has not been reported in Canada (1). Moreover, the virus has not been mechanically transferred.

Curly-top of geranium is of no economic importance in Ontario at the present time and no cases of this disease have been brought to my attention since the original one in 1958.

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LES PETALES VERTS DU FRAISIER ET LA PHYLLODIE DU TREFLE LADINO[1]

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Deux maladies d'apparition plutôt récente retiennent de plus en plus l'attention des pathologistes de l'Est du Canada. Ce sont les pétales verts du fraisier et le déclin virulent du trèfle Ladino. Ces deux maladies selon Frazier & Posnette (2) sont causées par le même virus.

La maladie des pétales verts, décrite par Posnette (8) en 1953, fut observée au Canada par Gourley (3) en Nouvelle-Ecosse deux ans plus tard. Depuis on l'a signalée dans Nouveau-Brunwick en Colombie Britannique au Québec et en Ontario (1, 6, 4, 9).

La maladie des pétales verts est probablement la plus récente des maladies du fraisier énumérées dans les revues de Plakidas (7) et de Mellor et Fitzpatrick (5). Cette maladie est caractérisée par la couleur verdâtre des pétales qui sont plus petits que des pétales normaux. Quelquefois les pétales sont légèrement teintés de rouge vers la fin de la floraison. Les fraisiers malades sont plus petits que des fraisiers sains et leur taille, considérablement réduite en certains cas, rend difficile le dépistage des individus malades dans un matelas fourni d'individus sains, à moins qu'il n'y ait un groupe compact de plantes malades. Quelques fleurs restent stériles mais ce n'est pas la règle générale; les ovules se développent sur le receptacle qui mûrit ratement, mais demeure vert et sec; les akènes sont proéminents au lieu d'être inclus dans le receptacle. Il arrive qu'une partie du receptacle mûrisse mais l'apex est toujours sec, dure et déformé.

Les vieilles feuilles sont vert-olive, jaunissent graduellement et tournent au rouge avant de sécher et mourir. Les feuilles qui se forment une fois que la plante est malade sont petites légèrement concaves avec marge vert-pâle et pétiole court. Les stolons, sont plutôt courts, si toutefois ils s'en forment.

Les symptômes du déclin du trèfle Ladino sont analogues, à ceux des pétales verts sauf que au lieu de virescense des pétales il s'agit de phyllodie, i.e. d'une transformation des organes floraux en organes foliaires. Toute la plante devient naine, les feuilles jaunissent puis rougissent. A ce stade de développement, il n'y a plus regénérescence par les stolons, et si, la plante ne meurt pas au cours de l'été elle ne survivra pas à l'hiver. De toute façon c'est l'hiver qu'on blâmera pour sa disparition.

Les enquêtes dans la province de Québec et les rapports provenant des autres provinces nous portent à croire que la maladie des pétales verts tend à augmenter avec les années. Au cours de ces enquêtes, on a observé que le pourcentage de plantes malades dans un champ dépend de l'âge de la plantation. Dans une fraisière en première année de production où le plant

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utilisé était certifié, on a rarement trouvé des fraisiers malades. Durant la deuxième année de production, on a observé des pourcentages de maladie allant jusqu'a 8%, alors que dans une fraisière en troisième année ce pourcentage a atteint facilement 20% et 25%.

Dans les pâturages le même phénomène se produit pour le trèfle Ladino. L'année d'établissement le trèfle est parfaitement sain et bien qu'il soit possible de voir des traces de phyllodie à l'automne de la première année de production, ce n'est qu'au cours de la deuxième année que la maladie prend des proportions alarmantes et, en troisième année, le trèfle Ladino disparait et cède la place aux gramiées.

Distribution géographiques

Les enquêtes que nous avons pu faire et les rapports déjà cités nous portent à croire que le développment de ces deux maladies est relié aux conditions climatiques. Ces maladies paraissent plus communes dans les régions du Bas- St-Laurent, du Lac St-Jean et de Québec, que dans la plaine de Montréal et les Cantons de l'Est, et parallèlement dans les Provinces Maritimes que dans le Sud-Ouest de la Province de Québec et l'Ontario où le climat est plus chaud.

Quelques données expérimentales

Au cours des deux dernières années, il a été possible de s'assurer qu'il s'agissait bien de maladies à virus grâce à des essais réussis de transmission au moyen de greffes. Nous n'avons pu toutefois réussir la transmission par la cuscute du fraisier au trèfle Ladino et réciproquement. Pour ces essais nous avons utilisé deux espèces de cuscute: Cuscuta gronovii et C. subinclusa.

Nous avons étudié la possibilité que ces deux maladies soient causées par le virus de la jaunisse de l'aster. Les essais de transmission de la jaunisse de l'aster par la ciccadelle de l'aster ont été infructueux pour provoquer la maladie des pétales verts et le déclin du trèfle Ladino. De même les tentatives pour provoquer la jaunisse de l'aster avec le virus des pétales verts et celui du déclin du trèfle n'ont donné que des résultats négatifs.

Des essais faits en plein champ laissent toutefois peu de doute que la maladie est disséminée par des insectes. En effect des parcelles de trèfle Ladino ont été semées en champ et une partie de celles-ci a été recouverte avec des cages de saran de 30 mailles au pouce carré et abondamment poudrée d'insecticide. Après trois années de croissance, nous n'avons pas observé de phyllodie à l'intérieur des cages alors qu'à l'extérieur cette maladie était très commune.

Il semble donc que ce virus ne puisse causer de ravages considérables dans les fraisières à cause de leur renouvellement régulier après deux années de production. Toutefois on considère déjà la déclin virulent comme un facteur limite de production du trèfle Ladino surtout dans les régions à climat frais.

Fleurs Avec Pétales Verts en Regard d'une Fleur Saine.

Trèfle Ladino Atteint de Déclin Virulent
et Montrant la Phyllodie des Capitules.

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A NEW HOST RECORD OF A RUST IN CANADA

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Uromyces cillarum (Grev.) Lév., a foliar rust of Spanish bluebell, Scilla hispanica Mill., was collected twice on Vancouver Island, B.C. in April, 1961; by the author at Victoria and by W.R. Orchard at Saanichton. A brief note of its occurrence is of interest as only two previous North American records exist. Arthur (1) lists a collection by Blasdale from Berkeley, California on S. hispanica and Savile (2) cites its occurrence on Hyacinthus sp. at or near Sidney, B.C.

Uromyces scillarum occurs widely in Europe and on the Mediterranean coast of Africa, and is known to occur naturally on various species of Bellevalia, Hyacinthus, Muscari and Scilla eastward to central Asia. It has been sparingly introduced into other parts of the world with horticultural stock.

The lesions on the bluebell specimens from Victoria occurred on both sides of the leaf. The chocolate-brown telial pustules were separate at first but later coalesced. Lesions were seldom found on petioles and never on the inflorescence or its stem. Both the foliage and flowers of infected plants were of normal size and vigor at the time of collection, but it is suspected that infected leaves would yellow and age earlier than normally. The Victoria specimens were collected in an old private garden planted about seventy years ago.

The occurrence of this microcyctic rust is more of academic than of economic interest.

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